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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 19 JANUARY, 1934 NUMBER 1

INFLAMMATORY TUMORS OF THE BRONCHI

EXPERIMENTAL AND PATHOLOGIC CONSIDERATION

ACHILLE PERONI, M.D.

MILAN, ITALY

Drs. Chevalier and Chevalier L. Jackson recently published an interesting paper on "Benign Tumors of the Trachea and Bronchi with Especial Reference to Tumor-Like Formations of Inflammatory Origin."[1] The article was based on the presentation of a number of clinical cases.

While the clinical and therapeutic aspects of these supposed inflammatory tumors are clear, there still remains some question as to their histologic classification and their etiology. Since these questions are of great importance, if not from the practical, at least from the academic, point of view, the Jacksons, during the preparation of their paper, asked me to see whether it was possible to resolve experimentally the question of the etiology of these tumors.

My researches gave negative results as to the possibility of reproducing experimentally a pathologic process in the bronchi that is akin to these inflammatory tumors, but as a result of histologic study, from the comparative point of view, of other types of inflammatory hyperplastic processes of the upper respiratory tract, I arrived at some conclusions on the question of the possible inflammatory origin of these tumors of the bronchi, as well as on the matter of their histologic classification.

It would be like starting to read a book from the conclusions to begin by discussing the naming of these tumors. In fact, the classification of something presupposes the knowledge of its intrinsic nature; for this reason, I shall return at the end of the article to the question of nomenclature. I shall use for the moment the provisory term "inflammatory tumors," which is that adopted by the Jacksons; these lesions are tumors in the physical sense of tumefactions, not in the sense of blastomas, and are inflammatory because inflammation is their most probable origin, or at least best explains their histologic and clinical character.

From the Chevalier Jackson Department of Bronchoscopic Research, Temple University School of Medicine, Philadelphia.

1. Jackson, Chevalier, and Jackson, Chevalier L.: Benign Tumors of the Trachea and Bronchi with Especial Reference to Tumor-Like Formations of Inflammatory Origin, J. A. M. A. **99**:1747 (Nov. 19) 1932.

PLAN OF THE WORK

A study of the etiology of inflammatory tumors of the bronchi may follow three directions: (1) an effort to obtain from the anamnesis of patients the possible cause of the formation of inflammatory tumors; (2) an effort to reproduce these tumors experimentally by reproducing in experimental animals those conditions which may be presumed to produce or favor the genesis of such lesions; (3) an effort to discover the etiologic explanation from a histologic study of the growths, especially by comparing them with other inflammatory or degenerative hyperplastic conditions in other organs.

ANAMNESTIC CONSIDERATIONS

A critical examination of the data of the anamnesis may throw much light on the question of the etiology of inflammatory tumors of the bronchi, and the consideration *post hoc ergo propter hoc,* though often wrong in the particular case, may be of exceptional value in the study of a series of cases.

A most important question to establish is whether or not the chronic inflammatory pulmonary and bronchial conditions that are often found in association with these inflammatory tumors are their consequence. It is almost the question of the origin of the egg and the hen, and there are enough good arguments in favor of both theories. Is it the purulent process in the lung or bronchus which produces the formation of the inflammatory tumor, or does this localized hyperplastic inflammation, by obstructing the drainage of the lung, favor the development of purulent bronchitis or of a pulmonary abscess?

One of the arguments in favor of the first supposition is that the stagnation of the purulent secretion produced by a chronic bronchitis, by a "bronchiectatic septic tank" or by an abscess of the lung constitutes the irritative factor which produces a *locus minoris resitentiae,* with the formation of hyperplastic inflammatory tissue. This argument has much value, and could be adapted to some cases but not to all. I am of the opinion that the irritation of the chronic suppuration may help the formation of granulation tissue in the bronchi, but this type of granulation tissue is quite different from the inflammatory tumors. I shall discuss in another part of this article the histologic difference between cases in which the granulation tissue is definitely of secondary origin and those in which it is apparently of primary origin. I shall say here only that granulation tissue depending on the irritating action produced by the traumatism of coughing and by the toxic action of pus comes from a bare stroma and is not covered by epithelium, while the inflammatory tumors lift the bronchial epithelium en masse and only in a second stage

undergo a metaplastic transformation. If these so-called inflammatory bronchial tumors were produced by the irritation from the stagnation of purulent secretion, they should be a much more frequent and common finding in cases of bronchiectasis and chronic abscess of the lung, which can be followed well during bronchoscopic treatment. However, I have observed bronchoscopically that these formations are rare, and that when the bacterial irritation and the mechanical traumatism from the purulent secretion produce a localized purulent inflammatory process of the bronchial walls with secondary formation of granulations, the latter are generally localized at points where the irritating actions are most marked, and especially on the bronchial spurs. These granulations, which are definitely of irritative origin, have a much more labile character than that of the inflammatory tumors; they are soft, flat and bloody, and as easily as they are produced they disappear by spontaneous resorption or by necrosis. As they are always flat, multiple and sessile, they cover the bronchial wall; they seldom produce phenomena of bronchial obstruction. If this type of inflammation causes obstruction, it is because the diffuse granulation of the orifice of a small bronchus causes an annular stenosis of its entrance, quite different from that produced by the inflammatory tumors.

Another argument in favor of the etiologic importance of the direct irritating action is the observation that as soon as the inflammatory tumor is removed, and with it the reason for the stagnation of secretion, the tumor has no tendency to recur. But this argument is a vicious circle. Chevalier Jackson has emphasized in many writings on various pathologic conditions of the lung the necessity of a free air passage and drainage of the secretions for the integrity of the lung and bronchus. He has demonstrated in a series of clinical cases that the bronchial obstruction, whether it is produced by foreign bodies or by malignant or benign tumors, may produce in the tributary bronchial and pulmonary fields, depending on the chronological epoch of the obstruction and on its "valvular type," more or less complete atelectasis, stoppage of the secretions with the formation of "drowned lung" and, when there is the action also of infectious processes, secondarily, bronchiectasis and abscesses of the lung. Many bronchial tumors which are benign from the histopathologic point of view are malignant clinically because of the suppurative complications that they produce. The association of malignant and benign tumors of the bronchi with bronchiectatic dilatations of the corresponding bronchial tree is a binomial accepted as a postulate by all pathologists. I have observed a large series of clinical cases which demonstrate that it is sufficient to remove the bronchial obstruction (foreign body or tumor) to see all purulent complications of the lungs and bronchi clear up.

All these arguments are theoretical, but when one enters the practical field one finds good arguments in favor of both theories. There are cases in which the history of the disease permits one to suppose that the suppurative process in the bronchi or in the lung is the causative factor in the production of the inflammatory tumor. There are other cases in which the bronchiectatic and suppurative processes seem to appear in a second stage, when symptoms of the presence of bronchial obstruction produced by the endobronchial tumor have already been present for a long time. I do not mean to say that in the latter cases there is no history of previous inflammatory processes and that the tumor had a sudden and inopinate origin like a blastoma, but simply that there are no evidences which permit one to think that the important factor was the irritation produced by coughing or by the purulent secretion coming from a preexistent bronchiectasis or pulmonary abscess. Of course, in the histories of the patients there are always some elements that suggest an inflammatory or degenerative origin of the tumor process. It is rather difficult to understand the cases in which symptoms of bronchial obstruction and of frequent hemoptysis appear in a patient with a past history that gives no cause for pathologic changes in the lungs. These patients have generally had one or more attacks of bronchopneumonia, in most cases of influenzal origin, which has generally not undergone a normal resolution.

The case of K. B. belongs to the group of cases in which it is possible that the irritative origin lay in a chronic purulent process; in fact, in the history of the patient the first symptoms were those of a productive cough which originated after an attack of grip, with a clinical diagnosis of chronic bronchitis and incipient bronchiectasis. The finding of a tumor in the right main bronchus partially occluding its lumen was quite accidental. The best indication that the chronic purulent bronchiectasis was an etiologic factor in the origin of the inflammatory tumor is that on bronchoscopic examination the suppurative process seemed to affect both bronchi.

The case of S. L. is similar. It was that of a woman who for nineteen years had frequent and copious purulent expectoration. Three months before she was examined bronchoscopically, she started to have dyspnea with blood-streaked expectoration. There were clinical signs of an abscess of the middle lobe, a diagnosis which was confirmed by bronchoscopic examination. There was also a mass of granulation tissue obstructing in part the entrance of the middle lobe bronchus, but in this case the removal of granulation tissue did not improve the condition very much.

In the case of MacL., there had existed for four years a purulent and hemorrhagic secretion, with the presumption of an abscess of the

inferior lobe of the left side; bronchoscopically, a pedunculated tumor was found occluding the left bronchus almost completely. From the details of the history of the patient and from the effects of the removal of the tumor, the growth was much more likely a secondary than a primary condition.

In some cases, like that of A. M., the initial etiologic factor is evident, the cause of the tumor-like granulation tissue in this case being a chicken bone lodged in the left main bronchus which produced an abscess of the left inferior lobe.

In other cases the etiology is complicated. E. H., when she was 17 years old, suffered from an abscess of the lung following tonsillectomy, which healed spontaneously, but when she was 31 years old dyspnea occurred, with frequent hemoptysis and elevation of temperature. At bronchoscopic examination, a tumor almost completely occluding the right main bronchus was found, with purulent bronchitis and atelectasis of the right lower lobe.

E. T. had for two years progressive dyspnea, with signs of atelectasis of the inferior lobe on the right side, and repeated hemoptysis. At bronchoscopy there was found a polyp of the right bronchus, which was removed, but recurred and reproduced the subjective symptoms and the physical signs of atelectasis. In this case the patient had had, during youth, many attacks of bronchopneumonia, of which the last one occurred four years before the appearance of the symptoms of the bronchial tumor.

In the case of E. G., that of a girl, aged 15 years, there was a history of a bronchopneumonic process of the influenzal type, antedating the "present illness" by a number of years. About six months before, she had cough, profuse expectoration and all the signs of atelectasis of the right lower lobe. On bronchoscopy, there was found a polypoid mass occluding the inferior lobe bronchus. The tumor was removed, and the atelectasis disappeared. A few months later, however, a reformation of the tumor occurred and a reproduction of the atelectasis, which again disappeared after the removal of the second tumor.

The case of J. D. P. was that of a man, aged 62, who six years before had influenza and bronchopneumonia of the influenzal type. Shortly before coming under observation he began to have hemoptysis and dyspnea, which were produced by the presence of partial atelectasis of the middle and inferior lobes of the right lung. The reason for the atelectasis was a tumor which occluded almost completely the right bronchus just below the bifurcation of the upper lobe bronchus. As soon as the tumor was removed, all of the symptoms disappeared.

If one examines the cases described by different authors and diagnosed as bronchial tumors of inflammatory origin, one finds that the concomitant pathologic condition of the lungs is more often of the secondary than of the primary type. The case described for the first time by Aucoin, and referred to also by Jackson, was that of a woman who nine years before had influenza, and after a few years had little by little noted the presence of asthmatoid breathing, so marked that at one time the diagnosis of asthma had been made, and later that of pulmonary tuberculosis. The explanation of this symptom was found in a tumor partially occluding the right main bronchus. All of the symptoms disappeared after the removal of the inflammatory tumor.

In the case reported by Burrel and Trail there was atelectasis produced by a tumor which disappeared after its removal. In the case reported by Kirsch and in that reported by Knock there was a bronchiectasis in the region of the bronchial tree distal to the tumor. In Ashbury's and Jesberg's cases the cough and the intermittent atelectasis cleared up after removal of the tumor.

Yankauer reported a case of abscess of the lung in which a phrenicectomy was performed, but the abscess was cured only after bronchoscopic discovery and removal of an inflammatory tumor of the bronchus.

In the case reported by Wessler and Rabin there were hemoptysis and atelectasis of the middle lobe of the right side; these symptoms appeared nine months after the patient had had bronchopneumonia of influenzal origin. The pathologic process was produced by an inflammatory tumor occluding the entrance of the upper lobe bronchus, and all of the symptoms disappeared after its removal. In another case described by the same authors there were a productive cough and atelectasis of the inferior lobe of the right lung, which were cured by removal of the inflammatory tumor of the bronchus.

In Zinn's case, a tumor supposed to be a fibroma, but probably the same type of inflammatory tumor as that being discussed, produced a cough and atelectasis of the inferior lobe of the right side. All of the symptoms disappeared after removal of the tumor.

EXPERIMENTAL STUDY

As I believed that these "tumors" of the bronchi are not neoplastic processes, but rather phenomena of localized hyperplastic inflammation, specific or nonspecific, depending on bacterial, chemical or physical factors, I tried to reproduce the same conditions in animals. For this research I chose the dog, an animal particularly favorable for endoscopic purposes.

In man it is easy to observe localized or diffuse inflammatory conditions of the mucosa of the respiratory tract caused by irritant chemi-

cal agents (war gases), gas or irritating dusts produced in factories, acids (in cases of vegetal foreign body), physical agents (localized traumatism produced by the presence of a foreign body or by the action of surgical instruments in the attempts to remove the foreign body) or infectious bacterial agents (suppurative diseases of the lungs and bronchi).

The inhalation of irritating gases and suppurative diseases of the lungs and bronchi generally have the tendency to produce diffuse inflammation of the mucous membrane of the tracheal and bronchial walls. Sometimes, in cases of abscess of the lung or purulent bronchitis, one may find inflammatory stenosis of the bronchial orifices, with the presence of granulation tissue. Much more frequently there is granulation tissue produced by a foreign body, either by mechanical irritating action (pins, tacks, bones) or by chemical irritation produced in the case of certain kinds of vegetal foreign bodies (above all, peanuts, which by fermentation produce a large amount of fatty acids).

In a few cases one may observe manifestations of bacterial inflammation localized to only one point on the mucous membrane, as in the cases in which a fistula forms from an infected mediastinal gland into the trachea or bronchi. In these cases the presence of granulation tissue around the fistula is easily seen both in the diffuse inflammatory processes and in the localized ones. Whether for chemical, physical or bacterial reasons, granulation tissue is always of a character quite different from that of the tumors which I am discussing; granulating fungations are shallow, sessile, multiple, bloody, soft and easily subjected to spontaneous necrosis or regression when the irritating cause ceases.

Since my attempts to reproduce inflammatory tumors of the bronchi experimentally were unsuccessful, I do not think that it is necessary to reproduce here in extenso the protocols of the experiments performed on more than twenty-five dogs. I shall only summarize the results obtained by different methods of irritation and inflammation.

Experimental Infection.—It has been impossible to produce experimentally diffuse inflammatory processes of the type of chronic bronchitis and bronchiectasis. For this reason I attempted to produce localized infections of the bronchial walls and of the peribronchial tissues (because I suspected that in some cases inflammatory tissue in the bronchus may come from peribronchial tissue by a phenomenon of herniation). In the attempts to produce localized infection a special kind of bronchoscopic syringe (devised by Chevalier Jackson and built on the same principle as his forceps) was used to inject the infecting substances. This instrument has a delicate touch and permits one to inject or to aspirate a certain amount of liquid with perfect precision. There are two sizes, one with a capacity of 0.5 cc. and the other with a capacity of 1 cc.

For infective material I used a culture of hemolytic and nonhemolytic streptococci and Staphylococcus aureus and also bronchial secretion from the bronchial tree of patients with chronic pulmonary abscess and bronchiectasis.

With the infective material from human patients I never succeeded in producing a pathologic process in dogs. In cases in which broth culture was injected into the submucous stroma of the trachea or bronchi, after two days the development of a small abscess was generally noted at the point at which the injection was made. These small abscesses underwent spontaneous regression in a few days, and in a short time normal membrane covered the point at which the abscess had formed. In none of these cases was a tendency to the formation of granulation tissue noticed. In cases in which the infective liquid was injected as deep as 0.5 or 1 cm. into the peribronchial parenchyma of the lung, no pathologic reaction was noticed endoscopically. Probably the pulmonary tissue succeeded in battling alone against the localized infection, and if a small localized abscess did form, it was able to drain itself into a small bronchus.

It is possible that the reason for the failure of the experiments with localized infections lay in the kind of animal on which the experiments were performed. The dog is well adapted to the purpose of practice in bronchoscopy, but it is not considered by bacteriologists as favorable for the purpose of bacteriologic experiments. It would have been better, perhaps, to choose an animal like the rabbit, which is susceptible to infection with the bacteria that are pathogenic for man. Unfortunately, this animal, because of its small size, is not well adapted to endoscopic researches.

Chemical Irritation.—The difficulty in choosing the kind of chemical traumatism was that strongly reducing or oxidizing substances (like silver nitrate, phenol and chromic acid) could not be used; these substances produce a cellular necrosis, rather than an irritative condition, with hyperemia and proliferation of tissue but not leukocytosis or destruction of cells.

Following the suggestion of Dr. Livingston, professor of pharmacology at the Temple University School of Medicine, I used as irritating substances, croton oil, mustard oil and naphthalene, and as the excipient and vehicle, hydrous wool fat, which was capable not only of dissolving these substances, but also of keeping the irritating substance, which could not be easily expelled by the cough reflex or the ciliary action, in contact with the bronchial mucous membrane for a longer time. The mucous membrane was resistant against all these means of irritation. A few drops of croton oil are enough to produce a tremendous hyperemia of the intestinal mucous membrane, but with a

concentration of croton oil in hydrous wool fat of 20 per cent, hyperemia of the bronchial mucous membrane could not be produced. The same negative results were obtained with 10 per cent mustard oil. Naphthalene and betanaphthol were completely inactive. With previous traumatization to remove the mucous layer, so that the irritating substance could act on the submucous stroma, a mild degree of inflammation of the bronchial wall was produced, but if hyperproduction of the connective tissue was present, it was so slight that it was impossible to appreciate it endoscopically. In two cases in which croton oil 20 per cent and mustard oil 15 per cent were used, the animals died of bronchopneumonia following the aspiration of this substance into the small bronchi; but even in these cases there was no sign of severe reaction in the mucous membrane of the large bronchi.

Also, with the use of chemical traumatism it is difficult to control the irritating action so as to be able to produce only an action exciting proliferation without necrosis of the cells; chemical substances are either too energetic or too mild. Only a mild irritating substance, acting in the same place for a long time, could succeed in this purpose, but in this case the associated action of the cough reflex and the cilia protects the mucous membrane and the submucous stroma.

Physical Traumatism.—It is much more easy to produce granulation tissue in the bronchial wall through physical and, above all, mechanical traumatism than by means of bacterial infection or chemical irritation.

As methods of mechanical traumatism, curettage of the bronchial walls with the bronchoscopic curet of Myerson and scarification with the double-claw forceps of Jackson were used. After repeated mild traumatism performed at many sittings (usually five or six), always avoiding too strong action and trying not to remove the granulation tissue of recent formation, I succeeded in causing the formation of some granulation tissue. The granulation tissue obtained by this method had all the characteristics of that observed in cases of foreign bodies that remain in the bronchial tree for a long time, bleeding easily and showing a marked tendency to undergo necrosis; this disappeared quickly as soon as the mechanical irritation was suspended. In none of the ten dogs in which the production of granulation tissue was obtained did the tissue show any tendency to behave like inflammatory tumors of man.

During the course of the experiments I also used thermal traumatism, or diathermocoagulation, a means by which it is easy to obtain different degrees of local temperature of tissue and all degrees of reaction from simple hyperemia to rapid coagulation. With mild action negative results were obtained, but with high temperature obtained with 4 or 5 ma. necrosis of the mucous membrane and the submucous stroma occurred; after the escher was eliminated, a tendency to the formation

of granulation tissue was sometimes observed, which, however, showed a strong regressive tendency and was quickly absorbed. In only one dog, after severe cauterization of the orifice of a small bronchus, a cicatricial stenosis of this orifice was obtained, but without the formation of granulation tissue of the chronic type.

Conclusions.—This experimental research was undertaken with the purpose of producing inflammatory polypi of the bronchi, and in this respect the results were completely negative. Some conclusions can be drawn, however, as to how easily the bronchial mucous membrane recovers from lesions produced by chemical, physical or infectious traumatism or localized infections of the wall. Also, if granulation tissue is produced by prolonged mechanical traumatism, this tissue has a strong tendency to reabsorb itself at once when the irritation is stopped. These experiments give an idea of the facility with which mucous membrane reacts against every chemical, physical and infectious insult, and how easily recovery of the lesions is produced when the physiologic conditions which cooperate in its defense are present, namely, free drainage, ciliary activity, good aeration and the presence of a good cough reflex. I can say only that if the reasons for the formation of these bronchial polyps is a local irritating or infectious action, it is difficult to reproduce experimentally conditions that have probably acted for a number of months. The type of bronchial granulation tissue that was obtained in the bronchi of the dogs by means of physical irritation was much like that which is observed in man as a result of the prolonged presence of foreign bodies, or that caused by chronic purulent diseases of the lung.

PATHOLOGIC CHANGES

In order to establish the etiology and pathogenesis of these so-called tumors of the bronchi, it is interesting to make a histologic study of them, especially comparing them with the various types of hyperplastic inflammatory processes and principally with those of the upper respiratory tract (particularly those of the nose and larynx). Of interest also is the comparison of these supposed inflammatory tumors with the true inflammatory tissue that is encountered in the bronchi following the irritation produced by foreign bodies or by germs in the purulent diseases of the lungs, and that which is often present in the collateral inflammatory processes in cases of bronchial carcinoma.

I do not have much to say concerning the question of the gross pathologic changes in these endobronchial tumors which are supposed to be of inflammatory origin. At bronchoscopic examination they are not much different from other benign and malignant endobronchial tumors, nor is it possible from morphologic examinations to establish a diagnosis. The tumors are more or less reddish, with a velvety

appearance, and often with evident signs of superficial vessels, which may explain the frequency of hemoptysis among the subjective signs of the disease. Tumors of this kind are more often pedunculated, but sometimes they are sessile. The rounded, almost spherical, form, with only a small pedicle to keep the tumor attached, is typical. There are many possible reasons for this form, but it is typical of a soft tissue to take a ball-like form when it expands in a cavity. This is according to the same physical principle by which a drop of water attached to a smooth surface takes first a hemispheric form, and afterward little by little detaches itself from the surface to which it is attached by capillarity, becoming longer and longer, until finally it looks like a pear attached to the surface only by a long neck. The same principle explains why nasal polyps which extend from the ethmoid cells take the hemispheric form before assuming the long form. Also, laryngeal polyps, whether they arise from inflammatory or from degenerative hyperplastic processes, which are usually subepithelial, always occur as a herniation of the submucous stroma to the exterior and take on a nodular form; the same is true of all tumors, even malignant ones, that expand in a hollow cavity—benign and malignant polyps of the stomach, the intestines and the bladder and the hyperplastic processes in the posterior half of the inferior turbinate. Another reason for the tendency to assume the nodular spherical form which applies also to nasal and laryngeal polyps and the hyperplastic processes involving the posterior and the middle turbinates, is the vicious circle that is present when the pedicle of the tumor is strangled and becomes thinner, with greater and greater difficulty in the venous and lymphatic circulation and hence the tendency to stasis and to the production of dilatation of the vessels, which may assume the appearance of angiomas. Because of this stasis the migration of the lymph and serum from the vessels into the connective tissue is much easier, and the latter takes on an edematous aspect (the formation of serous cysts may also occur). The mechanical action produced by the current of air moving in the two directions has a marked effect, as do the elongation and shortening of the bronchi at each respiratory movement, which may produce a molding effect on the inflammatory hyperplastic tissue, like the pill-roller of the pharmacist.

The microscopic picture of these tumors is especially characteristic at the first biopsy when one succeeds in taking out the tumor in toto, or when a large piece near the implantation can be removed. When, instead, biopsy specimens have repeatedly been taken from the tumor, it loses much of its original aspect and because of its tendency to recur and to have secondary inflammatory processes on its denuded surface it will be covered by recent granulation tissue mixed with coagulated

blood and made up of a few young fibroblasts in coagulated fibrin, with many cells of inflammatory origin.

In the slides in which the section has included the complete tumor, the covering epithelium is generally well preserved and continuously attached to the epithelium of the normal bronchial wall. On looking at the epithelium, one gains the definite impression that the mass of the tumor originates from the submucous stroma of the peribronchial tissue, and that the bronchial epithelium is lifted by the hyperplastic mass as the skin of the inguinal region is lifted by a hernia. The epithelium is mostly of the flat stratified type, but in some of the cases observed (for instance, that of E. H.) it was possible to observe easily between one fungation and another of the tumors that the epithelial covering in the protected introflexions had the original cylindric aspect, and still had well preserved cilia, while, in approaching the free surface of the tumor, it was possible to follow the modification of the epithelium that lost its cilia, becoming cuboid and finally flat (fig. 1). This metaplastic transformation of the endobronchial epithelium which covers these tumors is undoubtedly produced by the continuous traumatism of cough, of the secretions, of the respiratory movements of the bronchial walls and even of the current of air. The tumors are much more exposed to all the physiologic and pathologic traumatisms because of their position in the bronchial lumen.

What is really typical and different for different types of inflammatory tumors is the stroma. I do not think that it is necessary to give a detailed description of all the cases observed, but there are about three classic types. All of the cases observed by me and those described by various authors may be divided into these three classes, in which all the differences are in the stroma and depend on the type of connective tissue of which the stroma is built, as well as on the characteristics of the vessels. The distinction that I have made is not peculiar to the endobronchial tumors, but is based largely on the study of histologic pictures of hyperplastic and degenerative processes in the upper respiratory tract, which an eye trained to observe pathologic processes of the nose and larynx does not hesitate to find similar to these lesions in the bronchi. The Jacksons have given the detailed pathologic description of four cases in their recent article.[1]

Sometimes the stroma is of the type of fibrous connective tissue, with the same appearance that is noted in polyps of the larynx, which many pathologists diagnose as fibromas. The nuclei in this connective tissue are generally small, while the intercellular fibers are large and stain heavily with eosin; a few inflammatory infiltrations formed by a few lymphocytes and plasma cells are found, generally localized near the epithelium; the vessels are usually small and are circumscribed by stronger connective tissue (fig. 1).

There is another type with few cellular elements, mostly small nuclei, but with a great amount of intercellular substance, mostly serous, and frequent cystic formation; it does not take the eosin stain very well. In these cases the collagenous fibers are thin and much inflated by plasma; the mobile elements of chronic inflammation are more frequent, and the vessels are a little larger than in the previous type and are generally circumscribed by perivascular infiltration. These formations are much like the polyps of the nose which originate from chronic purulent inflammation of the ethmoid and the maxillary antrum, and also from the primary degenerative processes of the mucous membrane of the nose

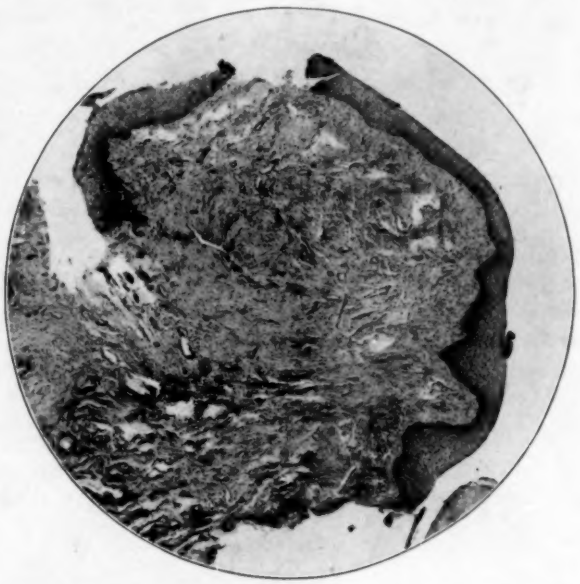

Fig. 1.—Inflammatory polyp of the bronchus of the fibromatous type.

and sinuses often found in allergic patients who suffer from asthma and paroxysmal rhinitis. The inflammatory and degenerative processes of this type are classified by many pathologists as myxomas, fibromyxomas or myxomatous polyps of the nose (fig. 2).

In the types of bronchial polyps formed from edematous connective tissue, there are often marked manifestations of stasis in the blood vessels and the lymphatic spaces, and the polyp with large dilated vessels is circumscribed by a ring of stronger connective tissue. These ectasias are often so large that the picture is like that of a cavernous angioma. This type of polyp of the bronchus is similar to a common kind of polyp of the larynx which many pathologists consider as a real neo-

Fig. 2.—Inflammatory tumor of the bronchus of the edematous type rich in capillaries. It is possible to observe that the epithelial covering still has its cylindric aspect in the introflexion, but the free surface is covered by flat metaplastic epithelium.

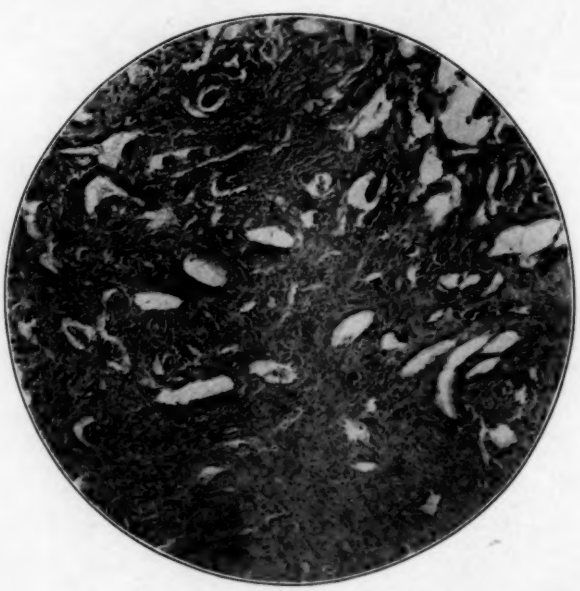

Fig. 3.—Inflammatory tumor of the bronchus of the fibro-angiomatous type.

plastic formation and which is called an angioma or a fibro-angioma. Other pathologists, including Dr. Konzelmann, do not believe in the hyperplastic origin of these formations in the larynx, and are much more inclined to consider the formation as a process of organization of an old hematoma, which hypothesis could be valuable in explaining the sudden appearance of these tumors on cords of quite normal appearance (fig. 3).

Sometimes these three classic aspects are modified by the taking of many biopsy specimens, which produce easy bleeding, organization of the clotted blood attached to the tumor and excitation of the proliferation of the stroma exposed through the opening of the thick epithelial capsule which protected it. In such cases there is a tumor characterized by the absence of epithelium, and constituted by a mass in which there are a few young fibroblasts constituting a sort of net which includes a large amount of fibrin in the process of organization, with a large amount of mobile inflammatory elements, leukocytes, lymphocytes, plasma cells and many long cells with pyknotic nuclei. In this type of inflammatory tumor it is common to observe elements having a large cytoplasm and clear nucleus, with an epithelioid appearance, and isolated or more often grouped together with a tendency to form alveoli. Some of these elements are similar to endothelium (in fact, they start to build the endothelium of new vessels); others seem to be rather of the reticulo-endothelial system, and probably fall short of the formation of fibroblasts by only one step. Many of these young cells have been regarded by Dr. Konzelmann as similar to the epithelial elements which arise from regenerating alveolar epithelium, which are found mixed in the young connective tissue which fills the alveoli in cases of splenization of the lung and postpneumonic and postinfluenzal or postatelectatic carnification of the lung and which show a tendency to the regeneration of the epithelial alveolar elements.

Sometimes these elements have a typical epithelial appearance and group themselves to form alveoli of the glandular type, showing a tendency to the regeneration of the bronchial glands. Such newly formed glands were found in one case in great abundance, in a tumor of the fibrous edematous type (A. R.) with a few manifestations of karyokinesis, but with no tendency to aggressive growth into the connective tissue. In this case it was difficult to decide whether there was only a regeneration of some glandular elements enclosed in an inflammatory tumor, or whether the lesion was a real benign adenoma (a type of tumor often described as common in the lumen of the bronchi) or a carcinoma with a low degree of malignancy. Very often one finds in the literature on the bronchi that the tumors have the histologic diagnosis of adenoma, but if one looks at the reproductions of the biopsy

specimens, one finds that probably too much importance has been given to a few epithelial elements enclosed in large masses of connective tissue of the fibrous or myxomatous type, probably of inflammatory origin, which is the real first element of the tumor.

In summary, the principal characteristic of these formations of the bronchi with a real neoplastic character is the presence of the typical stroma formed by connective tissue that is more or less fibrous or edematous and more or less rich in blood vessels, with the presence of very few inflammatory elements, and covered by a complete layer of epithelium which is generally flat and stratified, but is the metaplastic continuation of the ciliated bronchial epithelium.

The histologic structure of the tissues composing the bleeding granulations which are often found near foreign bodies of long standing and which may also be found in cases of pulmonary and bronchial suppuration, such as chronic pulmonary abscess and bronchiectasis, is quite different. I have already mentioned that granulations of this kind have a tendency to spontaneous regression or necrosis and do not represent anything so substantial as the inflammatory tumors. When this type of tissue is examined histologically, it is generally found attached to masses of clotted blood or fibrin in which one sometimes finds the entrance of new fibroblasts, as in an attempt at organization, these cells being attached to a mass of tissue of a typical inflammatory character, in which one sees all the elements of recent inflammation and very few young elements of the connective tissue. Between the masses of coagulated blood and fibrin, which are also in part necrotic, one sees leukocytes, lymphocytes and, above all, large mononuclear cells. There are also a few plasmocytes and fibroblasts with large nuclei, which attempt to form a connective tissue net. There are very few new capillaries. Altogether there is a predominance of the mobile elements, and this may explain the tendency to reabsorption and to spontaneous necrosis. There is an absolute lack of a regular epithelial covering, and only at the edges of the granulation tissue does one sometimes see an attempt at regeneration on the part of the bronchial epithelium.

The behavior of the connective tissue in cases of endobronchial malignant tumor is interesting. It is notorious that carcinoma of the lung is most often bronchiogenic, which does not mean, however, that it originates from the epithelium of the main bronchi. Generally, the lesion seems to be a real carcinoma of the lung because it takes origin from the epithelium of the small bronchi, and that is easily understood because the surface of epithelium lining the small bronchi is a hundred times larger than the total surface of the main bronchial epithelium; there is, therefore, much more chance of carcinoma originating from

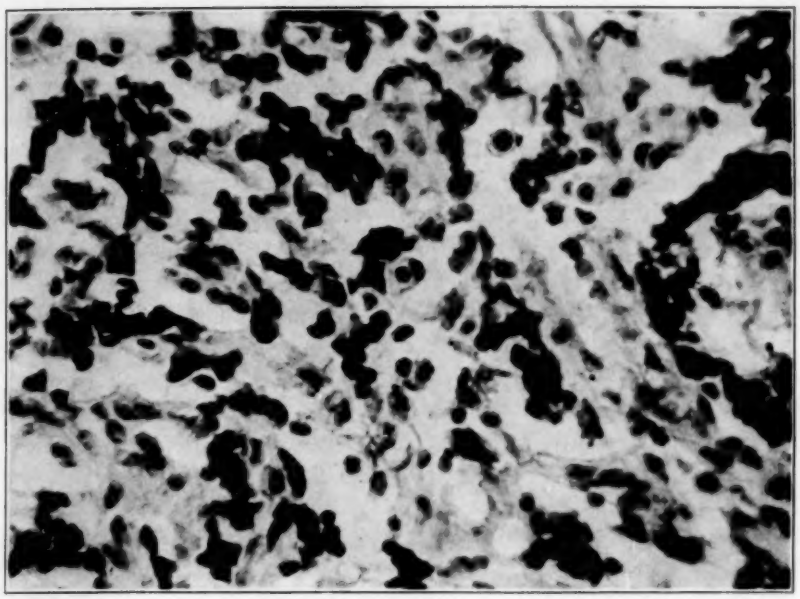

Fig. 4.—Inflammatory tumor of the bronchus as it appeared after many biopsy specimens had been taken. Note the young regenerating connective tissue.

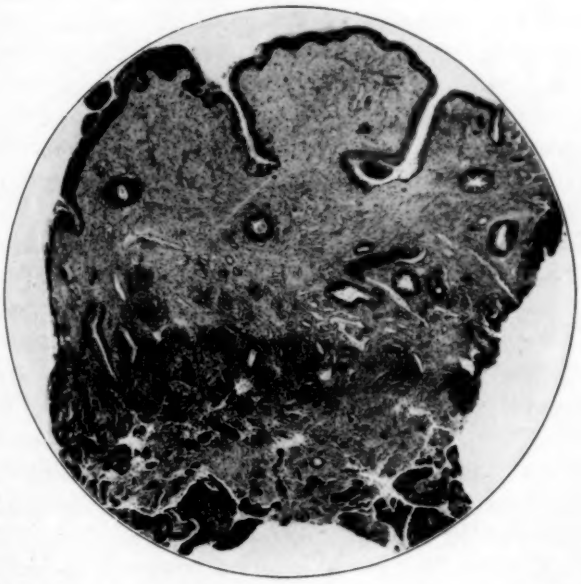

Fig. 5.—Carcinoma of the bronchus herniating into the lumen of the bronchus. It is easy to see that the greater part of the tumor was composed of fibro-edematous tissue of the inflammatory type; only near the pedicle of the tumor can a few islands of carcinomatous elements be seen.

the epithelium of the small bronchi. Bronchial carcinomas show a strong tendency to invade the parenchyma of the lung, and even when they take their origin from the epithelium of the main bronchi, they have much more tendency to intrapulmonary expansion than in invasion of the bronchial lumen. In cases of origin from the epithelium of both small and main bronchi, these carcinomas have a strange tendency to localize near the hilus. Often it is not until the second stage that there is a development of the neoplastic mass in the lumen of the large bronchi, and even when the tumor takes origin from the epithelium of the main bronchi, the growing into the bronchial lumen generally starts very late, when the mass has already reached large dimensions. When the tumor has expanded into the bronchial lumen, it is easy to take biopsy specimens endoscopically. Often the histologic material taken out of the bronchial lumen is composed only of connective tissue of the inflammatory hyperplastic type. If one succeeds in demonstrating the presence of neoplastic elements, they are generally buried by a large amount of connective tissue of the hyperplastic inflammatory type, covered by the bronchial epithelium which was lifted by the endobronchial mass (fig. 5).

This herniation of the neoplastic mass, together with a large amount of connective tissue, often forms a characteristic histologic picture, which, with the exception of the presence of blastomatous elements, recalls the aspect of the inflammatory endobronchial tumors and also of other hyperplastic degenerative processes of the upper respiratory tract, such as nasal polyps or laryngeal polyps. In such cases the neoplastic elements are found only near the base of attachment. All the other parts of the tumor are composed of connective tissue, mostly loose and deep-staining. Ectatic capillaries are frequent. These lesions are covered by an epithelium which is mostly flat and stratified. There are seldom inflammatory manifestations; if present, they are mild. The picture of the connective tissue surrounding the endobronchial carcinoma is similar to that of the hyperplastic and degenerative conditions of the upper respiratory tract which receive different names; those in the nose are called fibromyxomatous polyps, and those in the larynx, fibromas and fibro-angiomas.

It is easy to reconstruct mentally the genesis of this special kind of endobronchial tumor. In its expansive tendency the carcinoma grows at the same time as the connective tissue which surrounds it and tries to circumscribe it; when the tumoral mass reaches the bronchial lumen and herniates into it, the connective tissue, which is no longer limited and compressed by the surrounding tissue, has the tendency to assume the edematous type, and in this it is helped by the phenomena of stasis that are present when the pedicle of the attachment of the endobronchial

tumor becomes thinner. There is again a vicious circle, according to which the more the tumor grows, the greater is the accentuation of the phenomena of stasis in the vessels, with ectasia of the capillaries and myxomatous degeneration of the connective tissue. For these reasons, there is a larger growth of the connective tissue than of the neoplastic portions of the tumor.

Conclusions.—If the histologic observations in cases of endobronchial inflammatory processes are reviewed, two different types are seen. One represents something that may be called the superficial soft granulation; it is easily bleeding, falling into necrosis, and is composed principally of mobile elements of the inflammatory type with the characteristics of the granulation tissue found in pyogenic processes. This kind of granulation has no expansive tendencies of the tumor type, and does not ordinarily produce phenomena of endobronchial obstruction; in other words, it is not a real tumor. It is the type of granulation that is found in the chronic inflammatory processes of the lung and the bronchi and in cases of long-standing bronchial foreign bodies.

However, the endobronchial tumors not of the blastomatous type described by the Jacksons in their monograph [1] have a histologic picture that may be called subepithelial. In these cases the epithelium is integral in mass, and its only metaplastic manifestation is a tendency to thicken. In these cases the main stroma is predominantly of the connective type, with a fibrous myxomatous tendency, or characterized by vascular enlargements. In this connective tissue there is almost no sign of what may be the old inflammatory origin of the tumor; there are no signs of regression or involution; the entire tumor has a permanent character.

For any one familiar with the pathologic histology of the upper respiratory tract it is inevitable to compare mentally the histologic picture of these endobronchial tumors with the familiar ones of the hyperplastic processes of the nose, sinuses and larynx which have the clinical denominations of nasal and laryngeal polyps. The superior and the inferior respiratory tracts have a tendency to present, for different pathologic reasons, an especial pathologic condition that with different minor variations is always of the type of hyperplasia of the connective tissue, with more or less participation of the vessels. These hyperplastic conditions, in which the epithelium has only a passive participation, often have an inflammatory origin, as is the case in polyps of the nose and sinuses following chronic ethmoiditis and antritis; likewise, in the larynx, when the whole cord is in a condition of chronic inflammation, the polyp may be said to have an inflammatory origin. However, in cases of polyp of the nose in patients with asthma, the origin may almost surely be degenerative. In consequence of the repeated hyperemic condition of the mucous membrane of the nose

and sinuses following allergic sensitization, there are hyperplasia of the submucous connective tissue, stasis in the vessels and secondary edematous transformation of the connective tissue. In the case of laryngeal polyps, the denomination of inflammatory hyperplasia is still under discussion; in fact, some claim that mechanical herniation of the submucous connective tissue occurs following sudden vocal traumatism. I had occasion to examine histologically a cord with a polyp attached, and I was able to observe that in the tissue of the cord surrounding the pedicle of the tumor there was no sign of inflammatory reaction, and the submucous connective tissue of the cord continued into the polyp, with the modification that the same vessels seen on the cord seemed to be more ectatic in the polyp. For these reasons I insist on my impression, and I confess that it is only a question of induction because I have no histologic documentation, that the genesis of these tumors of the bronchi of connective tissue origin is either in the submucous stroma of the bronchus or, more probably, in the peribronchial tissue. It is possible that the origin is in old inflammatory processes and secondary organization with proliferative changes in the tissue of inflammatory origin, but nothing permits the exclusion of the possibility that phenomena of noninfective hyperemia may produce the same hyperplastic tendencies in the connective tissue. As for the question of origin of these proliferative nuclei of the connective tissue, they may be really bronchial, owing to an old bronchial inflammation, but they may also be the consequence of peribronchial proliferation. The fibrosis of old inflammatory processes of the pulmonary parenchyma, like the carnification of common type or influenzal bronchopneumonia, is common, with abnormal resolution or sequelae of partial atelectasis of the lung. Common also are the manifestations of connective tissue proliferation in the alveoli following inflammatory necrotizing inflammations of the lung (for instance, postdiphtheritic pneumonia), with complete destruction of the epithelium, and, as in those cases, one must think of the possible entrance of this net formed of connective tissue in the lumen of the small bronchi, with the formation of the typical pathologic process called chronic bronchiolitis obliterans. There is no reason to deny that this connective tissue, still intrapulmonary, may go into the bronchial lumen through the space between the incomplete bronchial cartilaginous rings. As a result of the obstruction that may occur to the return circulation in the herniated tumor, there may be enlargement of the vessels and edematous changes into connective tissue of vascular origin. The explanation given by Konzelmann, that the epithelial elements in some cases of inflammatory tumors of the bronchi may come from regenerating epithelium, may be a factor in this explanation. It is interesting that all patients with bronchial inflammatory

tumors have a complicated history from the point of view of previous pulmonary disease. Almost all of the patients studied had, two or more years before the onset of the symptoms, repeated attacks of bronchopneumonia, generally of influenzal origin. This coincidence of the history of poorly resolved processes of influenzal bronchopneumonic type in the history of patients with inflammatory tumors of the bronchi the Jacksons have emphasized strongly in their article, and it is known that influenzal bronchopneumonia is one condition which often produces phenomena of pulmonary carnification as a complication.

Classification.—Having expounded the morphologic character of these tumors, I shall try to draw some conclusions with regard to their classification. They are undoubtedly tumors from the clinical point of view, because they tumesce into the bronchial lumen, but I do not mean by the word tumors that they are blastomas, because they have no sign of atypical proliferation.

The denominations adopted by different authors for similar lesions are granulomas or inflammatory polyps. To adopt this last denomination would not differentiate these tumors enough from those formations which are more fungating, more bleeding and easily necrotizing, without any epithelial covering, which are really formed by recent granulation tissue without any tumoral appearance and which are much more like granulomas of other regions of the body. Many other authors have called these tumors fibromas; but in using this name one would fall into the same error as when this term was used in cases of polyps of the larynx, because in both cases one uses for hyperplastic conditions of connective tissue a denomination that should be reserved for true benign blastomas. For the same reasons, it would be erroneous to use for other types the terms fibromyxoma or fibro-angioma.

If one continues to identify these tumor formations in the bronchi with the similar hyperplastic and degenerative processes in the nose and larynx, generally of inflammatory and degenerative origin, definite terms cannot be used until pathologists and laryngologists have reached an agreement on a pathologic classification of the processes that clinically and indefinitely are called polyps of the nose and larynx, and until denominations like myxomas, fibromas and fibro-angiomas have been dropped for names that are better adapted to the clinical and pathologic character of the lesions. For the moment I believe that the term polyp adapts itself very well to the gross morphologic character, and by adding the adjective inflammatory one could specify that the process is not a blastomatous one, but that chronic inflammation is its most probable origin.

COMMENT

The main purpose of this work was to try to reproduce experimentally those special tumors of the bronchi that are not real blastomas, but of which the histologic picture is similar to that of certain other hyperplastic processes, like polyps of the nose and of the larynx, and which, because of their morphologic characteristics and clinical course, are supposed to be of inflammatory origin. With the means I had at my disposal to produce chronic inflammation of the bronchial walls, I succeeded in producing only the type of inflammatory tissue that is easily found in cases of chronic pulmonary or bronchial suppuration or in the presence of foreign bodies. From the results of the experiments with regard to the histologic character of the tumors which represent a definite entity and may be easily differentiated from the common granulation tissue of the bronchi and from the anamnesis of the patients, I do not believe that these tumors are in most cases produced by the irritating action of the pus coming from suppurative processes of the bronchi, though in rare instances this may be a cause. From the observation of cases and in accord with the principle regulating the general pathology of the lungs, namely, that every obstacle to the free drainage of the bronchi and the air passages favors the genesis of suppurative processes of the corresponding pulmonary and bronchial field, I am inclined to believe that the bronchiectasis and suppurative diseases of the lung that often are seen associated with these tumors of the bronchi are more rationally to be regarded as secondary to the tumor obstruction. Both from the anamnesis of the patients and from the morphologic characteristics of the lesions, especially in comparison with those of the hyperplastic inflammatory processes involving the nose and throat and those of the connective tissue that surrounds bronchial carcinomas, these lesions of the bronchi with tumor form seem to consist of subepithelial hyperplastic tissue, probably of inflammatory origin, which may represent a herniation of chronic inflammatory tissue coming from carnification processes of the lung into the bronchial lumen. Most of the patients, in fact, give a remote history of frequent bronchopneumonia, usually of influenzal origin, which has run an atypical course and resulted in partial carnification of the pulmonary tissue.

Until pathologists have reached an accord on the denomination of these tumor-like inflammatory lesions of the respiratory tract, one cannot accept for them or for polyps of the nose or of the larynx such names as fibromas, fibro-angiomas and fibromyxomas, which should be reserved for real blastomatous processes. For the present the only rational denomination with which to qualify the gross appearance of the growths and their probable origin is inflammatory polyps of the bronchi.

IMPORTANCE OF ROENTGENOLOGIC EXAMINATION OF THE SINUSES IN CHRONIC ARTHRITIS

WITH SPECIAL REFERENCE TO CASES IN WHICH THE SINUSES ARE A SILENT FOCUS OF INFECTION

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There is a marked divergence of opinion in medical literature as to the etiologic relationship of sinusitis to chronic arthritis. The late Dr. John E. MacKenty [1] wrote: "I have considerable proof in my own records to offer in support of my contention that sinusitis and arthritis are closely associated." Hurd,[2] Faulkner,[3] Craig,[4] Burgess [5] and Kistner [6] hold similar views. On the other hand, Wood,[7] Mullin [8] and Anderson [9] hold the opposite view and claim that sinusitis is but infrequently or rarely of etiologic significance in chronic arthritis.

The present report covers a five year period of study from 1926 to 1931. A detailed stereoscopic roentgenologic study of the sinuses was made as a routine measure in 386 cases of chronic arthritis seen in office practice in the city of New York. The combined clinical and

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roentgenologic study in these cases has convinced us that paranasal sinusitis played a most important rôle in the progress and prognosis, and perhaps even in the onset, of the chronic arthritis of many of our patients. In many of the cases the removal of other foci of infection, or of the absorption of toxin had no effect, or, at most, only ameliorated the arthritis but did not result in a clinical cure, whereas the institution of rhinologic treatment of the sinuses was followed in many instances by an immediate and unmistakable improvement of the arthritic symptoms. In most instances the rapidity and degree of improvement seemed to bear no characteristic relationship to the degree of involvement of the sinuses or to the type of rhinologic therapy instituted. Some of the patients showed extensive and marked roentgenologic changes in the sinuses but comparatively little rhinologically. In some cases conservative rhinologic treatment, such as washing out of pus from the antrums, resulted in immediate marked improvement. In others in which the arthritis failed to show sufficient response to the conservative form of rhinologic therapy, more radical procedures, such as exenteration of the ethmoid cells or of the antral mucosa or opening and drainage of the sphenoid sinuses, were required before a satisfactory clinical improvement of the arthritis could be obtained.

The antrums and ethmoid sinuses were most frequently involved. Disease of the sphenoid sinuses had been most frequently overlooked, probably because of the greater difficulty in demonstrating disease in the sphenoid sinuses both clinically and roentgenologically.

In some of the cases the onset of sinusitis preceded the onset of arthritis. In the majority of the cases, the time relationship between the onset of sinusitis and the onset of arthritis could not be determined for the reason that in the majority (83 per cent) of the cases of infected sinuses the sinusitis was of a "silent" nature, and neither the patients nor their physicians had been aware of its existence.

For the purposes of this study no attempt was made to group the cases according to any of the many detailed classifications of arthritis offered by various students and writers on the subject. The 386 cases studied do not include the specific forms of arthritis caused by syphilis, gonorrhea, tuberculosis, gout and other specific infections or pathologic conditions. The term "chronic arthritis" as used in this paper applies, with the foregoing exceptions, to a clinical entity of more than six months' duration, in which the patient complains of painful or limited movement, or of both, involving one or more joints. Some of the patients had been almost totally incapacitated for many years. The histories of some of the cases in which excellent results were achieved dated back as many as ten to tweny years. In view of the fact that a clinical cure of the arthritis has persisted for from three to five

years in some of the most chronic and most incapacitating cases in this group and that recurrences of indeterminate origin were few, it is felt that the clinical cure in these cases is due mainly to the rhinologic treatment instituted rather than to any spontaneous remissions of the disease. It is worthy of note that 23 patients whose roentgenologic and rhinologic examination revealed sinusitis, but who refused to submit to rhinologic treatment, showed no clinical improvement or grew progressively worse in spite of all other treatment instituted. But at the same time, it must also be pointed out that in 19 patients who showed evidence of sinusitis clinical improvement resulted without resort to treatment of the sinuses. In these cases, the elimination of the other etiologic factors seemed to be sufficiently effective for the clinical control of the disease process.

The age of the patients varied from 15 to 77 years. One hundred and forty-two cases occurred in men and 244 in women. The majority of cases occurred in the third, fourth and fifth decade.

Of 93 cases in which sinusitis was demonstrable roentgenologically as well as by rhinologic examination, roentgenograms were made of one or more joints in 75 cases. In these cases, roentgenologic evidence of arthritis was demonstrable in 63 instances, or 84 per cent. The changes consisted of distinct narrowing of joint spaces, dislocations, lipping, condensation or rarefaction of the articulating bones, erosion of the surfaces of the joints and of destructive and hypertrophic changes in the bones. Frequently destructive as well as hypertrophic changes were noted in different joints of the same patient. Often both destructive and hypertrophic changes were noted in the same joint. Some of the most badly crippled patients showed little or no roentgenologic evidence of disease of the joints. It was not possible to classify this group of patients on the basis of correlated roentgenologic and clinical signs. Clinically, a careful study of the patients revealed, with but few exceptions, a probable focus of infection or a possible site of absorption of toxin somewhere in the body. In a few of the cases, metabolic disturbances seemed to be the major factor in the production of the chronic arthritis. But even in these cases, correction of the metabolic factors was usually not sufficient for the clinical cure of the patient unless simultaneous treatment was instituted against any infectious or toxic factors present. In all types of chronic arthritis, the possibility of an undiscovered focus of infection or absorption of toxin must be kept in mind, searched for and eradicated, if possible.

Roentgenography of the sinuses of the entire group of 386 patients was carried out in a single x-ray laboratory under the personal supervision of one of us (Dr. Fineman). This was of considerable practical importance because the roentgenologic interpretation of sinusitis

depends largely on the establishment of a standard type of roentgenography.

In the 386 cases, the sinuses looked entirely clear in 124 instances, or 32 per cent. In 262 cases, or 68 per cent, the roentgenograms revealed slight, moderate or marked changes, suggestive or indicative of sinusitis. The changes were of slight degree in 126 cases, of moderate degree in 102 and of marked degree in 34. The degree of arthritis bore no relationship to the degree of involvement of the sinuses.

Of the total group, 126 patients were referred to rhinologists for a clinical examination. In 33 of the cases with positive roentgenologic indications, the rhinologist could not find corroborative evidence of active disease of the sinuses. In 93 cases, the rhinologists' reports stated that the clinical examination substantiated the roentgenologic signs. In other words, in 3 of 4 cases with positive roentgenologic signs the rhinologist was able to demonstrate corroborative evidence of sinusitis.

It is difficult to understand the marked differences of opinion held by writers in different sections of the country as to the etiologic significance of sinusitis in chronic arthritis. Perhaps the etiologic factors vary in importance in different groups of cases. Age, economic status, race, physique and previous and coexistent disease all possibly modify the course of the disease and its response to treatment. Climatic conditions undoubtedly govern, to a marked degree, the incidence of sinusitis in chronic arthritis. Sinusitis is known to be extremely prevalent in New York and its vicinity. This probably accounts for the large incidence and the greater etiologic importance of sinusitis in chronic arthritis in patients seen in New York and the adjacent parts of the Atlantic seaboard. Similarly, the comparatively small incidence of sinusitis in the inland states, and particularly in such arid areas as Arizona, New Mexico and parts of California, would explain the lesser frequency and lesser significance of this disease in the cases of chronic arthritis seen there.

Admittedly there are many cases of paranasal sinusitis in which arthritis never develops. But similarly, there are many persons who harbor for years dental, tonsillar, prostatic and other infectious processes who do not develop arthritis, while others with the same types of infection do develop arthritis.

We believe that a demonstration of the relationship of sinusitis to arthritis depends not only on showing a large incidence of sinusitis in arthritis, but also on a demonstration of the improvement or cure of the patient following treatment of the sinusitis.

These postulates have been met by the demonstration of the existence of infection of the sinuses in a large percentage of our cases of

chronic arthritis and by the excellent clinical results which followed the rhinologic treatment in these cases. In 5 of the 51 patients treated rhinologically, the unsatisfactory end-results were considered to have been due largely to lack of cooperation on the part of the patients. Of 46, 28 received conservative rhinologic treatment such as nasal medication, suction, packs, nasal and sinus irrigations and operations on the septum. Of these 28, there was no improvement of the arthritis in 1. Of the remaining 27, 7 were moderately improved, 10 showed marked improvement and 10 others very marked improvement. Eighteen patients submitted to more radical procedures, such as radical operations on the antrum, ethmoidectomies and enlargement of the openings into the sphenoid sinuses. In this group, 1 patient failed to show any improvement, while of the remaining 17, 1 showed moderate improvement, 5 marked improvement and 11 very marked improvement.

We do not claim that sinusitis is of etiologic importance in the majority of cases of chronic arthritis. In view of the fact that in 83 per cent of the cases studied the sinusitis was of the silent type, its existence being unknown both to the patient and to his physician, and since the exact time relationship between the onset of sinusitis and arthritis was unknown to most of the other patients, it is obvious that clinically, at least, a cause and effect relationship between sinusitis and chronic arthritis could not be established.

However, clinical observation of the patients in this series clearly showed that rhinologic treatment instituted on the discovery of diseased sinuses had a direct and markedly beneficial effect on the arthritis of many of the patients thus treated.

The present routine roentgenologic study of the sinuses was begun about five and one-half years ago following the accidental discovery of a case of silent sinusitis in an arthritic patient (fig. 1). The successful demonstration of diseased sinuses in the arthritic patient depends on the understanding and avoidance of certain technical roentgenographic errors, as well as on the appreciation of a number of clinical points commonly overlooked.

It is not generally appreciated that roentgenography of the sinuses presents marked technical difficulties and numerous pitfalls, which lead to erroneous interpretation of the roentgenograms and which probably account for the generally expressed dissatisfaction of the rhinologist and clinician with the roentgenologic diagnosis of sinusitis. This is particularly true in the cases in which the pathologic process in the sinuses produces only slight variations from the normal in roentgenograms of the sinuses.

A detailed technical discussion of all the possible technical errors in roentgenography of the sinuses would be out of place here. Reference

must be made, however, to a few factors which are the essentials of diagnostic roentgenography of the sinuses and which should be understood by the rhinologist and clinician. In brief they are:

(*a*) The films must exhibit the greatest possible contrast between the air-containing sinuses and their surrounding structures. It is this quality which determines, to a large extent, the diagnostic value of the roentgenogram of the nasal sinuses. The quality of the roentgen ray which will produce the best contrast on the films must be ascertained in every case and for the various views of the sinuses by the tedious method of trial and error. This is a task which consumes a great deal of time and material and the importance of which is not fully understood or

Fig. 1.—Roentgenogram showing infection of the frontal sinus of the "silent" type in a case of chronic arthritis unsuspected clinically.

appreciated by many clinicians and rhinologists. In the arthritic patient with his deformities and slow and painful movements, the difficulties of the task are multiplied many times, but must be countered by the ingenuity and patience of the roentgenologist, if information of value is to be obtained from the roentgenologic examination.

When it is realized that a slight technical error may destroy on the film the roentgenologic evidence of disease in a sinus and that the successful clinical management of a case depends at times almost entirely on the ability to demonstrate roentgenologically this diseased sinus, it becomes obvious that every possible refinement of technic should be utilized in the roentgenologic examination of the nasal sinuses and that no amount of time and effort spent to attain this end should be considered unnecessary or superfluous. The technical difficulties and

pitfalls of roentgenography of the sinuses are stressed here because it is only through understanding and appreciating these that the roentgenologist can obtain the close cooperation of the clinician and rhinologist and through them the whole-hearted cooperation of the patient, a factor of great importance in these cases.

(*b*) On account of the marked complexity of the cranial structures and the complicated anatomic relationships of the nasal sinuses, stereoscopic films are of paramount importance and of utmost value in roentgenography of the sinuses and in diagnosis. Stereoscopic films enable the observer to eliminate confusing superimposed shadows of the cranial structures which are often so puzzling in the "flat" film. Furthermore, stereoscopic views are of the greatest importance to the surgeon in his study of the anatomy of the nose and sinuses prior to operation on them.

(*c*) Roentgenograms of the sinuses must be made in four or five positions. The object of this is to bring into view clearly and individually every sinus of both sides of the head. This point cannot be emphasized too strongly. Superimposition of the shadows of the sinuses on each other and of extraneous structures on the sinuses, slight rotation of the head on its vertical axis or slight movement of the head during the roentgen exposure destroys diagnostic detail. We have seen many cases in which, because of lack of attention to these points, definitely diseased sinuses were entirely overlooked.

It is apparent from the foregoing statements that a better appreciation by the clinician of the fundamental diagnostic qualities of the roentgenograms of the sinuses and of the necessity of a careful and detailed roentgenologic study of the sinuses would aid him in the management of many cases of chronic arthritis which prove to be refractory to the usual therapeutic measures.

The clinical search for evidence of sinusitis in chronic arthritis reveals the following types of cases:

(*a*) There is a certain number of patients whose history indicates clearly the presence of sinusitis. In the majority of these the roentgenologic and rhinologic demonstration of the infection is not difficult.

(*b*) There is a second group of patients who harbor an obscure low grade nasal infection which is usually overlooked because it is considered of insufficient importance for any detailed roentgenologic or rhinologic study. The history of these arthritic patients shows that their treatment had been thorough in all respects but this one, and that in spite of all treatment they had grown progressively worse. A cure can be effected only after a seemingly unimportant infection of the sinus is demonstrated and treated or eradicated.

(*c*) A third group of patients with arthritis in whom sinusitis is known to be present fails to show improvement in spite of attempted treatment of the sinuses because a roentgenologic study is usually done in a perfunctory routine manner, with the result that the roentgenograms of the sinuses are either misleading or incomplete and do not reveal the total extent of the pathologic involvement of the sinuses.

(*d*) A fourth group consists of patients in whom the history points to infection of the sinuses, but in whom sinusitis is entirely overlooked or only partially diagnosed because both the clinician and the rhinologist, relying entirely on transillumination and intranasal inspection, find nothing indicating infection and fail to avail themselves of the aid of a roentgenologic examination of the sinuses. Yet it is well known that the ethmoid cells and sphenoid sinuses cannot be transilluminated and are often even beyond the scope of intranasal or retropharyngeal inspection. Furthermore, in low grade catarrhal or polypoid disease of the sinuses, intranasal evidence of the disease process is usually slight or entirely lacking.[10]

(*e*) A fifth group of patients consists of those who suffer recurrences of arthritis in spite of what might be considered efficient treatment of the infected sinus. Such cases are cited by some as proof that chronic arthritis is an incurably progressive disease and that treatment of the infected sinuses of these patients has no effect on the ultimate course of the arthritis. Such argument does not take into consideration that these arthritic patients had probably developed the disease originally because of an impaired constitutional resistance to infection, and that they are particularly susceptible to new intercurrent infections in the sinuses as well as in other organs of the body. In these cases one must be on the constant watch for new infections in order to secure and maintain a permanent clinical cure of the patient.

(*f*) The sixth group of cases, which we have chosen to call the "silent sinus cases," is an important group and comprises a surprisingly large proportion of the cases of sinusitis. There is no history of sinusitis, yet the patients harbor definite infection of the sinuses, which is almost invariably overlooked because of the silent character of the infection. The occurrence of these cases makes the routine roentgenologic examination of the sinuses imperative in every case of chronic arthritis, regardless of the absence or presence of symptoms of sinusitis.

COMMENT

The many possibilities of error in the roentgenologic and clinical demonstration of infection in the sinuses show clearly how frequently

10. Looper, E. A.: Infection of the Nasal Accessory Sinuses, West Virginia M. J. **25**:710 (Dec.) 1929. Hurd, Lee: Personal communication to the authors.

the presence of sinusitis in a patient with arthritis can be overlooked unless the most painstaking efforts are made in the search for the infection. At the beginning of this paper it was stated that in a clinical study of this character, the mere demonstration of the frequent occurrence of sinusitis in arthritis would not prove the point that sinusitis is an important etiologic factor in arthritis. The clinical cure or improvement of the patient's arthritis following treatment or eradication of the sinusitis would be of far greater value in corroborating the thesis of this paper. A survey of the histories of the 368 patients with chronic arthritis has convinced us that many patients had failed to obtain relief either because infection of the sinuses had been entirely overlooked or because a faulty and incomplete roentgenologic examination of the sinuses had obscured the diagnosis, thus making it impossible for the rhinologist to eliminate an existing sinusitis as a focus of infection. Other patients had failed previously to obtain relief because, incredible as it may seem, they had had numerous operations on the sinuses without a single roentgenologic examination, so that the full extent of their infection had never been accurately determined.

The exceedingly difficult task of amelioration or eradication of disease of the sinuses cannot be overemphasized. It is a task which frequently taxes the clinical and surgical skill of the most experienced rhinologist. We are convinced, however, that treatment and surgical intervention undertaken only after a most thorough and complete clinical and roentgenologic study of the sinuses is of the utmost importance in the successful treatment of chronic arthritis. We feel that sinusitis played an important rôle in the onset and prognosis of many of our cases of arthritis, and that the excellent clinical results obtained in many of our patients must be attributed in large measure to the aid of the rhinologist. Some of the patients failed to respond to every other form of treatment, and the arthritis grew progressively worse until the infection in their sinuses was treated or cleared up. It was only then that the patients began to improve, and the arthritis began to respond to treatment. No case of chronic arthritis was observed in which it could be said that the arthritis was aggravated by rhinologic treatment of the patient's sinusitis.

We believe that we are not overemphasizing the relationship of sinusitis to chronic arthritis. Perhaps the incidence of such a relationship varies in different sections of the country in proportion to the general prevalence of sinusitis. In our experience with patients from New York and its vicinity, sinusitis was found to be too frequent and too important a factor to be disregarded. Even if the sinus infection could not be eradicated entirely, all efforts were directed toward building up the patients' general resistance and removing the infection to as great a degree as possible. If the cure of only a few cases, after all other therapeutic measures had failed, can be attributed directly to the

removal of infection of the sinuses, it seems that in refractory and progressive cases of chronic arthritis an unfavorable prognosis is never justified unless every possible effort has been made to rule out and remove or eradicate paranasal sinusitis as a focus of infection.

REPORT OF CASES

A few typical case histories are cited:

CASE 1.—Mrs. C. G. H., aged 50, was referred to this office by Dr. Lee Hurd on Nov. 30, 1925. She gave a history of arthritis of five years' duration, involving chiefly the knees and, to a lesser extent, the feet, back and fingers. She walked with difficulty and suffered many sleepless nights because of the pains in the joints. Seven years previously she had an attack of acute sinusitis which had since

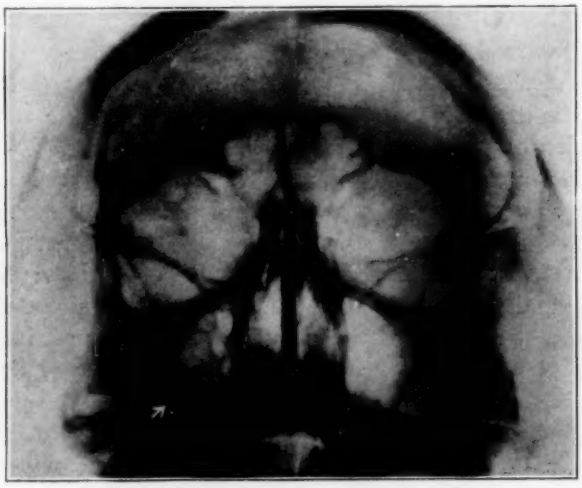

Fig. 2.—Roentgenogram showing moderate cloudiness and mucosal thickening in the right antrum in a case of chronic arthritis of five years' duration. Exenteration of the right ethmoid cells and a window operation on the right antrum were performed in 1925. There are excellent clinical results to date.

recurred three times. From the beginning she had been under the continuous and consecutive care of four prominent rhinologists in different parts of the country. After a tonsillectomy in 1920, the arthritis improved slightly. Her right antrum had been punctured and irrigated twenty times. Each antral irrigation was followed by slight temporary improvement of the arthritis. None of the rhinologists in the case had considered radical surgical measures on the sinuses of any value as a possible cure of the arthritis. Dr. Hurd, under whose care the patient was at the time of our examination, frankly acknowledged that he was dubious as to the effect of radical treatment of the sinuses on the arthritis. He felt that some other constitutional factor or focus was probably of greater importance in the case. With the exception of gastro-intestinal stasis and disease of the sinuses, our medical and roentgenologic examination revealed no foci or factors that could be considered of etiologic importance. The patient was consequently put on a corrective diet low in carbohydrates and was given a series of colonic irrigations,

baking of the joints, massage and vaccine therapy. After six months of this type of treatment the patient showed some improvement, but not of sufficient degree to warrant continuance of this method of therapy. It was then concluded that a radical operation on the sinuses offered the best chance of a cure of the arthritis, and Dr. Hurd was consequently prevailed on to undertake the operation. A roentgenologic examination of the sinuses revealed moderate cloudiness and thickening of the mucosa of the right antrum (fig. 2), as well as pathologic changes in the ethmoid cells. In May, 1926, a window operation on the right antrum and an exenteration of the right ethmoid cells were performed. There was an immediate and marked response in improvement of the arthritis. By November, 1926, the patient was practically free from all pain in the joints, and could walk up and down stairs without difficulty. When last seen, Dec. 23, 1931, there had been no recurrence of the arthritis.

CASE 2.—Mr. B. L., an orchestra conductor, aged 40, was examined on Jan. 12, 1931. He gave a history of the gradual onset of pain in the left shoulder. The pain had increased markedly during the previous two weeks and interfered with

Fig. 3.—*A*, roentgenogram showing pansinusitis of the silent type in a case of chronic arthritis. Cloudiness of the sinuses is most marked on the right side. Because of lack of rhinologic symptoms, great difficulty was experienced in persuading the patient to submit to a roentgenologic examination of the sinuses. There are excellent clinical results to date after exenteration of the ethmoid cells and window operations on both antrums. *B*, from the same case, showing cloudiness of the frontal and ethmoid sinuses.

his sleep. He felt fleeting pains in the other joints of the body and had lost 24 pounds (10.9 Kg.) in weight in the past six weeks. He had had a tonsillectomy eighteen months previously. Physical examination revealed extremely painful and limited motion of the left shoulder. Roentgenologic examination of the left shoulder revealed a calcific deposit just above the head of the humerus. There was nothing in the history suggestive of infection of the sinuses. With great difficulty, the patient was persuaded to submit to a roentgenologic examination of the sinuses. The roentgenograms of the sinuses revealed the presence of a pansinusitis (fig. 3). Dr. Lee Hurd's examination revealed marked deviation of the septum, a highly congested nasal mucosa and evidence of pathologic changes in all of the sinuses. An autogenous, Staphylococcus albus vaccine was made from the antral material. The intramuscular injection of 1 minim (0.06 cc.) of this vaccine containing 10,000,000 bacteria per cubic centimeter caused a "flare-up" of

every joint in the patient's body. After recovery from this acute attack, an exenteration of the ethmoid sinuses and a window operation on the antrums resulted in a complete clearing up of the arthritis within a few weeks. Since that time and up to March 31, 1932, there has been no recurrence, and the patient has been back at work as conductor of his orchestra.

Case 3.—Miss M. A. K., aged 29, was first examined on June 15, 1928. She gave a history of progressive arthritis of ten years' duration, involving both ankles, knees, hands, wrists and elbows. She was a telephone switchboard operator, but her fingers and arms had become so crippled that she had not been able to work for the past five years. She could walk for short distances with the aid of a cane. At the time of examination she was unable to dress or to feed herself. She had had a great deal of medical treatment without any effect on the arthritis. The disease was steadily progressing in spite of vaccine therapy, a tonsillectomy, and the removal of several abscessed teeth between the years 1921 and 1924. The

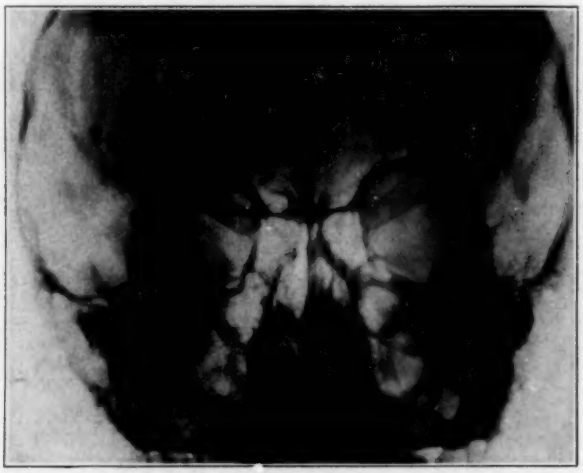

Fig. 4.—Roentgenogram showing slight cloudiness of the left ethmoid cells and crowding of the left nasal passages in a severe case of chronic arthritis. There is excellent clinical recovery to date, since an operation on the septum, exenteration of the left ethmoid cells and enlargement of the ostium of the left sphenoid in December, 1928. At operation, polypoid degeneration of the left ethmoid cells was found.

financial burden of all previous attempts at a cure and the progressive nature of the disease made the patient feel totally discouraged. Roentgenologic examination of the hands and left knee revealed marked bony changes. There was no history of sinusitis. Routine roentgenologic examination of the sinuses revealed, however, slight cloudiness of the right antrum and left ethmoid cells and crowding of the left nasal passages (fig. 4). After six months of active medical treatment there was only slight improvement of the arthritis. Rhinologic examination corroborating the roentgenologic signs revealed substantiating evidence of a low grade infection of the left ethmoid sinus. In this case again, because of the progressive nature of the disease and the resistance of the arthritis to every other form of treatment, it was decided that the sinuses were perhaps of greater importance as a focus of infection than the rhinologic and roentgenologic evidence

seemed to indicate, and radical treatment of the nose and sinuses was then urged on the rhinologist. In December, 1928, Dr. Lee Hurd performed a submucous resection and left ethmoidectomy and enlarged the opening into the left sphenoid sinus. By January, 1929, the arthritis showed marked improvement, and the patient discarded the use of the cane for walking. To date, the arthritis has shown progressive improvement. The patient walks as much as 1 or 2 miles a day, is able to dress and feed herself and has regained the use of her fingers to such an extent that for the past fifteen months she has been able to maintain uninterruptedly a job as telephone switchboard operator. This case is of particular interest because of lack of response to extraction of presumably infected teeth, tonsillectomy and vaccine therapy. The only definite response to therapy occurred following the operation on the sinuses. The patient is now happy, practically free from pains in the joints and economically independent, whereas for five years prior to the operation, she had been almost totally incapacitated and a burden to herself and her family.

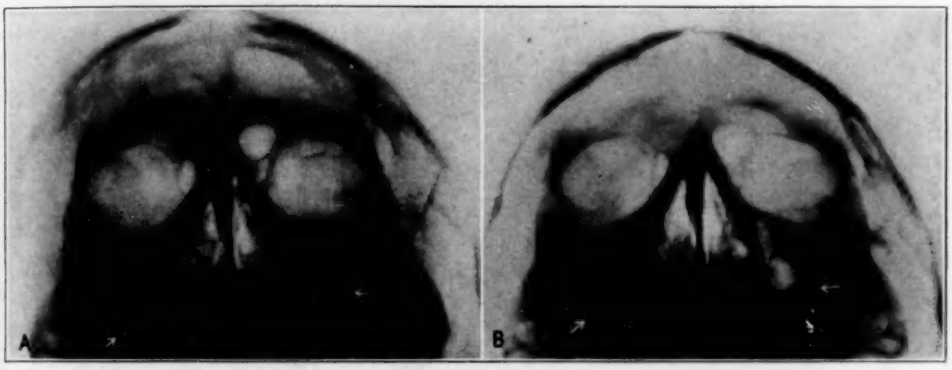

Fig. 5.—*A*, roentgenogram showing silent sinusitis in a case of chronic arthritis of one year's duration. Marked cloudiness of both antrums is shown. *B*, from same case was made with the patient in an upright position and reveals that the cloudiness of the left antrum is due to fluid within the sinus.

CASE 4.—Mrs. H. L., a housewife, aged 39, was first examined on March 26, 1931. She complained of pain in the left arm, left knee and left hip of one year's duration. In the last two months the pains had grown worse and were felt also in the left shoulder and across the small of the back. There was nothing in the history to suggest the presence of sinusitis. Nasal breathing was free. There was no antral tenderness, postnasal dripping or nasal discharge. When a roentgenologic examination of the sinuses was suggested to the patient, she objected strenuously to the procedure because of lack of any symptoms referable to the sinuses. When prevailed on finally to undergo the examination, the roentgenograms revealed fluid in the left antrum, a marked cloudiness of the right antrum (fig. 5) and cloudiness of the ethmoid cells on both sides, somewhat more prominent on the right. The turbinates looked hypertrophied, and the nasal passages appeared to be crowded. Dr. G. R. Brighton found a moderate amount of mucopus in both antrums. Antral washings and conservative treatment of the ethmoid cells were followed by complete loss of the patient's symptoms of arthritis. This

occurred in spite of the presence of definitely diseased tonsils, which were removed one year later merely as a prophylactic measure.

Case 5.—Mrs. E. O., aged 31, a housewife, was examined on Sept. 18, 1929. She complained of marked and progressive difficulty in chewing, of six months' duration. At the time of the examination she could hardly open her jaw on account of the marked pain in the temporomandibular joints. The left joint was more painful than the right. Eighteen months before she had had a tonsillectomy because of an attack of severe pain in the right shoulder. Six weeks prior to the examination she had had three teeth removed, without effect on the arthritis. There was nothing in the history suggesting sinusitis. A routine roentgenologic examination of the sinuses revealed marked cloudiness of the left antrum (fig. 6). Physical examination disclosed nothing of etiologic importance. Movement of the jaw was markedly limited, painful and accompanied by crackling and grating sounds over the left temporomandibular joint. On September 23, Dr. Hurd punc-

Fig. 6.—Roentgenogram showing marked cloudiness of the left antrum in a case of temporomandibular arthritis. There was nothing in the history to suggest sinusitis. There was complete recovery after a submucous operation and a window operation on the left antrum.

tured the left antrum, obtained pus and advised a radical operation on the antrum. Because of objection to this by the patient, it was decided to observe the effects of conservative treatment of the antrum. Until November, 1929, antral irrigations were resorted to with but slight relief of the pain in the jaw and with no improvement in the amount of mobility of the jaw. It then became apparent that the antral irrigations were insufficient for a cure, and Dr. Hurd's original suggestion, a submucous operation as well as a window operation on the left antrum, was then carried out. The operation was followed by immediate improvement. By July, 1930, the patient had gained 20 pounds (9 Kg.) in weight, could eat without any discomfort and could move her jaw freely. When last seen on Dec. 23, 1931, there had been no recurrence of the arthritis nor had any other joints become involved.

Case 6.—Mrs. T. F., a housewife, aged 50, was first seen on March 5, 1924, because of pain of six months' duration in the left shoulder, knees and neck. Roentgenograms of the knees revealed lipping of the patellae. Examination of the

sinuses at that time by Dr. J. King revealed no pathologic changes. There was no history of sinus infection. There was evidence of gingivitis and of considerable pericemental thickening around the roots of two pulpless teeth. Under general, dietetic and vaccine therapy, the patient's symptoms disappeared completely, and she remained well until December, 1928, when she returned for observation because of slight pains in the right elbow and neck. Roentgenograms of the cervical spine made on Dec. 5, 1928, revealed lipping of the bodies of the fifth and sixth vertebrae. Two devitalized teeth were still present but showed no evidence of the formation of an apical abscess. The roentgenologic examination of the sinuses revealed slight cloudiness of the right ethmoid cells, right antrum and right sphenoid sinus (fig. 7 *A*). A rhinologic examination and puncture of the right antrum did not reveal evidence of infection. Under general dietetic and vaccine therapy, the patient again improved and remained well until September, 1929. At that time she suffered a recurrence of arthritis in the neck and left shoulder. The pulpless teeth had not as yet been removed because of the patient's objection to this procedure. The teeth showed, however, no progressive changes roentgenologically.

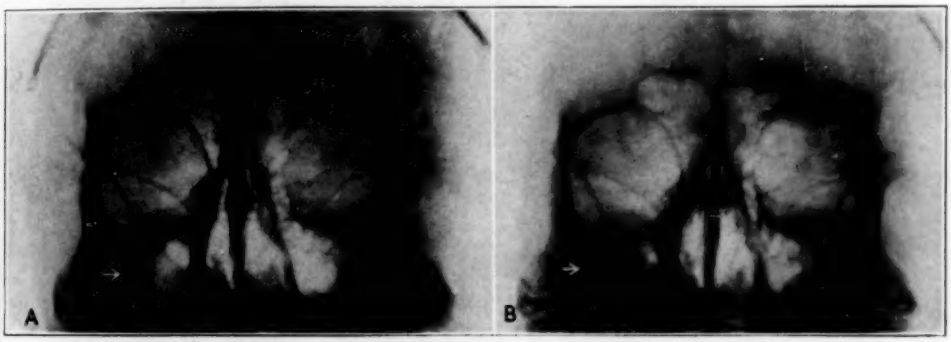

Fig. 7.—*A*, roentgenogram showing slight cloudiness of the right antrum in a case of chronic arthritis. Rhinologic examination failed to reveal clinical evidence of infection in the antrum. *B*, from the same case, was made eleven months later. A recurrence of the arthritis was found to be due to the development of an intercurrent infection in the right antrum. Pus was found in the right antrum. A short period of conservative rhinologic treatment resulted in a marked improvement in the condition of the patient.

In the search for a new focus of infection, the sinuses were reexamined, and a distinct and marked cloudiness of the right antrum was demonstrated (fig. 7 *B*). On puncturing the antrum, pus was obtained. After a short period of repeated antral lavage, the patient showed a marked improvement. When last seen on Dec. 23, 1931, there had not been any recurrence of the arthritis.

Case 7.—Mrs. R. A., a housewife, aged 41, was first examined on Sept. 26, 1929. She complained of severe pains in the elbows, hands and knees. The symptoms in the joints began several years previously. For the past three months she had been practically helpless and found great difficulty in getting about. She gave a history of sinusitis dating back many years, and was suffering from occipital headaches at the time of the examination. She had had a submucous resection in 1919, a radical operation on the left antrum and partial exenteration of the left ethmoid cells in 1927 and a radical operation on the right antrum nineteen months

prior to the present examination, and she had been dismissed by the rhinologist as "cured." The routine roentgenologic examination of the sinuses on September 27 revealed cloudiness of the ethmoid cells and blurring of the walls of the cells of both sides, moderate cloudiness of both antrums and moderate cloudiness of the left sphenoid sinus (fig. 8). Another possible focus of infection was in several devitalized teeth which did not show, however, the formation of an apical abscess. The tonsils had been cleanly removed in childhood. Rhinologic examination by Dr. Lee Hurd on September 28 revealed adhesions between the ethmoid cells and the septum, pus in the left antrum and pus coming from the left sphenoid sinus. On October 9, Dr. Hurd removed considerable scar tissue from in front of the left sphenoid sinus, enlarged the ostium and evacuated considerable pus from the sphenoid sinus. He also removed a portion of the wall of the left ethmoid sinus, some ethmoid cells along the orbit and some polypoid tissue from the left antrum.

Fig. 8.—Roentgenogram showing unsuspected infection of the left sphenoid sinus in a case of chronic arthritis. Free pus was found in the sphenoid sinus. Following drainage of pus, a left ethmoidectomy and removal of polypoid tissue from the left antrum, the patient made a rapid recovery.

Within seven weeks after the operation, the patient no longer suffered from her headaches, the nose had healed and the arthritis showed marked improvement. The patient has remained well to date.

SUMMARY

1. A routine stereoscopic roentgenologic study of the sinuses was made in 386 cases of chronic arthritis.

2. In 93 cases, sinusitis was proved to be present by a combined rhinologic and roentgenologic study of the sinuses.

3. Only 16 of the 93 patients were aware of the fact that they harbored disease of the sinuses.

4. In the remaining 77 patients, the sinusitis was of the silent or asymptomatic type.

5. Rhinologic treatment of these patients resulted almost invariably in marked clinical improvement of the arthritis.

6. The degree of arthritis bore no relationship to the degree of involvement of the sinuses. Some of the most brilliant clinical results were obtained in patients with advanced arthritis following rhinologic treatment of sinuses only slightly or moderately involved in a pathologic process.

7. Some of the patients responded to rhinologic treatment of a conservative nature, while in others, more radical procedures were necessary before a good clinical result was obtained.

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A NEW INTRANASAL OPERATION ON THE ETHMOID SINUS

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During the last three or more decades, several intranasal operative procedures have been described for the relief and cure of diseases of the ethmoid sinus. In general, these may be divided into two types: the radical and the conservative. In the radical operation (Hajek,[1] Mosher,[2] Sluder,[3] Ballenger,[4] Coffin[5]) the middle and superior turbinates and the anterior and posterior groups of ethmoid cells are removed. In the conservative operation (Pratt,[6] Halle[7]) part or all of the middle turbinate is preserved and the ethmoid cells lateral to these structures are dissected away. The favorable results in the latter type are probably due in a large measure to the fact that the nasal mucous membranes are preserved. In recent years, the tendency seems to be toward the more conservative types of surgical treatment.

It is not the purpose of this paper to discuss methods of diagnosis or the different pathologic types but to describe an entirely new and hitherto unreported method for the surgical care of the diseased ethmoid sinus. This operation is more radical than the Pratt or Halle procedures. It resembles the conservative and differs from the radical operations in that every effort is made to preserve the nasal mucous membranes. I have performed it in fifty-nine cases since January, 1932. There has been no serious untoward result in any case in my series.

From the Ear, Nose and Throat Department, Green's Eye Hospital.

1. Hajek, M.: Pathologie und Therapie der entzündlichen Erkrankungen der Nebenhohlen der Nase, ed. 5, Vienna, Franz Deuticke, 1926, p. 404.

2. Mosher, H. P.: The Surgery of the Ethmoidal Labyrinth, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1914, p. 232.

3. Sluder, Greenfield: A Surgical Consideration of the Upper Para-Nasal Cells, Tr. Am. Laryng. A., 1916, p. 200.

4. Ballenger, W. L.: Diseases of the Nose, Throat and Ear, Medical and Surgical, ed. 2, Philadelphia, Lea & Febiger, 1909, p. 233.

5. Coffin, Lewis A.: The Surgical Management of Nasal Accessory Sinus Disease, M. Rec. **81**:1021, 1912.

6. Pratt, Fred J., and Pratt, John A.: Intranasal Surgery, Philadelphia, F. A. Davis Company, 1924, p. 212. Pratt, John A.: The Present Status of the Intranasal Ethmoid Operation, Arch. Otolaryng. **1**:42 (Jan.) 1925.

7. Halle, M.: Die intranasalen Operationen bei eitrigen Erkrankungen der Nebenhohlen der Nase, Arch. f. Laryng. u. Rhin. **29**:73, 1915.

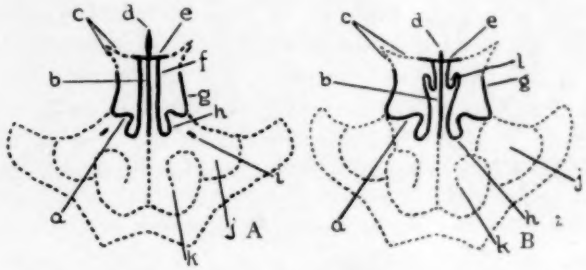

Fig. 1.—Scheme of the structure of the ethmoid bone (modified from the diagram in Hajek, Heitger and Hansell: Nasal Accessory Sinuses, vol. 1, p. 7). *A* represents the anterior ethmoid region; *B*, the posterior ethmoid region; *a*, the ethmoid bulla; *b*, the perpendicular plate; *c*, the frontal bone; *d*, the crista galli; *e*, the cribriform plate; *f*, the overhang of the middle turbinate; *g*, the paper plate of the ethmoid; *h*, the middle turbinate; *i*, the uncinate process; *j*, the maxillary sinus; *k*, the inferior turbinate, and *l*, the superior turbinate.

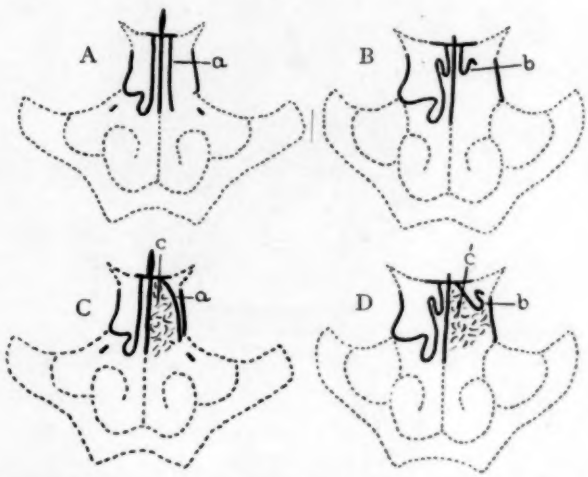

Fig. 2.—*A* represents the anterior ethmoid region (same as figure 1 *A*, after removal of the middle turbinate and ethmoid cells); *B*, the posterior ethmoid region (same as figure 1 *B*, after removal of the middle turbinate and ethmoid cells); *C*, the anterior ethmoid region (same as figure 2 *A*, showing the mucous membrane of the overhang of the middle turbinate held against the lateral wall of the nose by loose gauze packing); *D*, the posterior ethmoid region (same as figure 2 *B*, showing the superior turbinate fractured laterally and held in place by loose gauze packing); *a*, the mucous membrane of the overhang of the middle turbinate; *b*, the superior turbinate; *c*, gauze packing.

In order to operate with the greatest degree of safety, the patient should be in the upright position, as recommended by Mosher.[8] By this means, the backward biting into the ethmoid labyrinth is more apt to occur in a direction almost parallel to the floor of the anterior fossa. If the patient is lying down, the biting and pressure of the instruments are more likely to take place directly against the floor of the anterior fossa, which increases the danger of perforation, with possible injury to

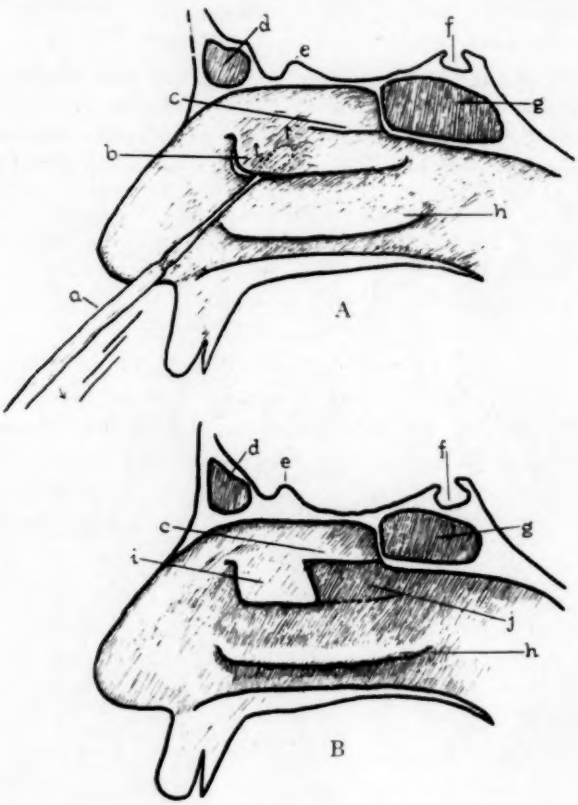

Fig. 3.—*A* represents the lateral wall of the nose, showing submucous resection of the anterior end of the middle turbinate with the Freer septal elevator; *B*, the completed operation; *a*, the Freer septal elevator; *b*, the middle turbinate; *c*, the superior turbinate; *d*, the frontal sinus; *e*, the crista galli; *f*, the pituitary fossa; *g*, the sphenoid sinus; *h*, the inferior turbinate; *i*, the mucous membrane of the overhang of the middle turbinate, and *j*, the portion of the middle turbinate removed.

the meninges and the substance of the brain. For this reason, the upright position offers an important factor of safety which must be considered.

8. Mosher, H. P.: Arch. f. Laryng. u. Rhin. **29**:245, 1915.

If the following type of anesthesia is used, a satisfactory view and a relatively bloodless operative field are obtained in most cases. The patient is given a preliminary dose of sodium ethyl barbiturate (from 3 to 4½ grains [0.2 to 0.25 Gm.]) thirty minutes before being sent to the operating room. The preliminary use of a sedative allays any feeling of apprehension that the patient may have and lessens the danger of several of the unpleasant sequelae (fainting, hysteria, collapse) for which the local anesthetic is usually blamed. The mucous membranes are shrunk with tightly wound cotton on the end of wire applicators which have been dipped into a solution of equal parts of 20 per cent cocaine and 1 : 1,000 epinephrine hydrochloride. When the mucous membranes have become thoroughly blanched, long cotton tampons moistened with the same solution are placed along the nasal chink from the choanal rim forward to the anterior ethmoid region. Two or more of these

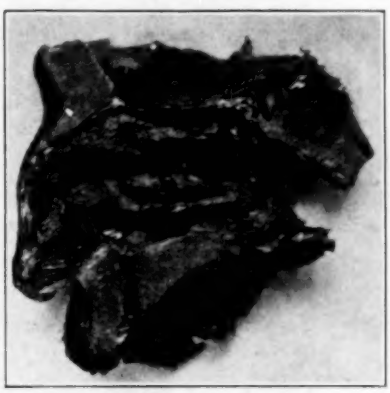

Fig. 4.—Photograph of the completed operation.

tampons are then placed in the middle meatus throughout its entire length. An additional one is placed over the region of the agger nasi in preference to an injection of procaine hydrochloride. The tampons are left in place for twenty minutes. I have never used "cocaine mud" because of the danger of overdosage,[9] although it is often used by many excellent operators. If the amount of epinephrine recommended is used, the field is relatively bloodless and the necessity for placing gauze or cotton tampons in the nasopharynx is obviated.

In most cases, a preliminary submucous resection is advisable in order to obtain more room and provide a better view for the ethmoid dissection. If polypi are present, they must be removed at this time. When this has been done, a linear incison is made along the anterior

9. McGuigan, H. A.: A Text-Book of Pharmacology and Therapeutics, Philadelphia, W. B. Saunders Company, 1928, p. 375.

half of the lower border of the middle turbinate, and a submucous resection (fig. 3 *A*) of this structure is performed with a Freer septal elevator. This mucous membrane and the superior turbinate are carefully preserved. The remainder of the middle turbinate is then amputated throughout its length with a pair of curved nasal scissors. The Bulla cells are next broken into by an ethmoid forceps (that devised by Breuning, Skillern or Wilde), and the dissection is carried backward by successive bites until the anterior wall of the sphenoid sinus is reached. The remainder of the dissection of the ethmoid sinus is then continued by working carefully upward until the solid bone separating the nose from the anterior fossa is reached and cleared of bony partitions and sinus mucous membrane. Likewise, all the partitions are broken down laterally until the lamina papyracea is reached and cleared throughout its length. Great care must be taken in the dissection of the superior and lateral portions of the posterior ethmoid region to avoid injury to the optic nerve. The agger cells are curetted away by means of a Pratt ethmoid curet.[6] Halle's [7] method of turning down the flap of mucous membrane in the agger region and chiseling away the cells should be used when there is considerable hypertrophy of the agger cells and the uncinate process. Loose spicules of bone and débris are carefully removed by the use of nasal forceps or by wiping with cotton swabs. When any septal deflection has been corrected and the bony part of the middle turbinate and the ethmoid cells removed, an excellent view of the interior of the ethmoid capsule is obtained. The superior turbinate and the mucosal covering of the middle turbinate have been carefully preserved (fig. 3 *B*). A nasal forceps is now placed between the septum and the superior turbinate, and the latter is fractured laterally (fig. 2 *D*). Iodoform gauze or gauze containing compound tincture of benzoin is now loosely placed in the space between the septum and the medial wall of the ethmoid capsule, in order to hold the latter in place. This is removed in twenty-four hours (fig. 2, *C* and *D*).

The postoperative care consists in the use of mild antiseptic mentholated oily drops after the removal of the gauze. Care must be taken that synechiae are not allowed to form between the septum and the mucous membrane of the medial wall of the ethmoid capsule. These structures soon become adherent to the upper and lateral walls of the nose, providing most of that region with a normal mucous membrane covering.

In this operation, most of the favorable features of the Pratt [6] and Halle [7] operations have been preserved (greater safety during the operation and preservation of the sense of smell by not injuring the olfactory nerves and their dural sheaths). The nose is normally moist afterward,

is not adversely affected by cold weather and shows little or no tendency toward the formation of crusts (provided the dissection has been thorough and no other sinuses are involved). Likewise, there is seldom any complaint of stuffiness in the nose, which one occasionally encounters after the more conservative procedures when a hypertrophic, bullous type of middle turbinate has been left intact. The method has the additional advantage of providing more room and consequently allowing better visual control at all stages than is possible in the conservative operations.

In approximately one of every four cases in my series, the superior turbinate underwent tremendous hypertrophy and filled a large part of the newly created space in the upper part of the nose.[10] I have seen it enlarge to such an extent that it approximated the size of a normal middle turbinate within three months.

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10. Pratt, John A.: Compensatory Nasal Growths, Ann. Otol., Rhin. & Laryng. **32**:211, 1923.

INFLUENCE OF "EXCITEMENT" ON THE DURATION OF POSTROTATIONAL NYSTAGMUS

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During the course of an experiment which has been fully described in an earlier paper,[1] certain observations were made which had no particular relevancy to the problem then under investigation but which were of such a striking and perplexing nature as to demand independent consideration. A group of ten adult common pigeons, which were to be subjected to repeated bodily rotation in an attempt to reduce the duration of their postrotational nystagmus through habituation, were tested on two successive days for nystagmus by means of twenty turns in the clockwise direction at the rate of one turn in 1.5 seconds. Since the average duration of the nystagmus for the group on the first day was 21.7 seconds and on the second day, 18.5 seconds, there was thus recorded an average reduction of 3.2 seconds. In the light of previous observations, which had indicated that an appreciable reduction in the duration of nystagmus is always to be expected between two successive tests under the conditions just described, this average reduction of 3.2 seconds was regarded as entirely natural and quantitatively usual.

On the following day, which marked the beginning of a twenty-one day habituation period, each bird was subjected to twenty 30 second periods of rotation such as had been employed on the two preceding test days, with rest periods—also of 30 seconds' duration—interpolated between the successive rotation periods. Following the initial trial on the first day of the habituation period, the group showed an average duration of nystagmus of only 14.2 seconds,[2] which represented a decrease of 4.3 seconds in comparison with the average of the preceding day. This was contrary to what might have been safely predicted on the basis of earlier observations, which had always indicated that when birds are tested on successive days, the greatest reduction occurs between the first and second day. However, since all of the average

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From Northwestern University.

1. Fearing, F., and Mowrer, O. H.: The Effect of General Anaesthesia upon the Experimental Reduction of Vestibular Nystagmus, J. Gen. Psychol., to be published.

2. During the nineteen succeeding trials, the average nystagmus time dropped to 9.7 seconds.

values obtained on the three successive days possessed a fairly low statistical reliability, it was merely assumed that the greater reduction observed in this particular instance between the second and third day (4.3 seconds) than between the first and second day (3.2 seconds) was due to chance variables, and no further attention was given to the matter at that time.

Following the initial rotation on the last day of the habituation period, the average duration of the nystagmus was 7.5 seconds, and during the succeeding nineteen rotation periods, it ranged between 7.8 and 6.7 seconds, thereby indicating that the average duration of the nystagmus for the group had reached a relatively stable level (a sort of "physiologic limit") and that continuation of the repeated rotation would probably be unproductive of any further decrease.

On the two days following the habituation period, the birds were tested in exactly the same way as before the repeated rotation had been commenced. The average duration of the nystagmus on the first day of the retest was 8 seconds and on the second day, 8.3 seconds. Although the difference between the average duration of the nystagmus following the initial rotation on the last day of the habituation period (7.5 seconds) and that obtained on the two retest days was not reliable statistically, it was sufficiently conspicuous to direct attention again to the more striking difference previously noted between the average duration of nystagmus on the two original tests and that obtained following the initial rotation on the first day of the habituation period.

It has already been implied that the speed, duration and other physical characteristics of the rotation used for both the original and the final tests were identical with those of the rotation used during the habituation period. The birds were likewise immobilized (by being wrapped with a narrow strip of cloth) and hooded in exactly the same manner at all times. It was therefore not easy to determine the cause of the relatively longer duration of nystagmus recorded on the original and final test days than on the initial trials on the first and last days of the habituation period. The only variation in procedure which could be recalled consisted in this: On the test days each bird was taken from its cage, immobilized, hooded and immediately placed on the rotator and tested, while still somewhat excited in consequence of having been caught and handled; whereas, on the habituation days each bird (except the first one [3]) was taken from its cage and immobilized and hooded while the preceding bird was being subjected to the first few of the twenty successive periods of rotation. This meant that during the habituation period the birds were usually wrapped and hooded and

3. The bird which happened to be selected first was, of course, wrapped, hooded and tested immediately on being taken from its cage.

allowed to rest quietly and undisturbed for ten or fifteen minutes before being placed on the rotator and given the initial twenty turns.

In order to determine whether this presumably inconsequential difference in procedure could, by any remote possibility, be responsible for the observed difference in the duration of the nystagmus on the test and habituation days, the same ten birds were subjected to the following routine: Every second day the birds were tested for nystagmus exactly as they had been on the two days preceding and the two days following the habituation period. On the intervening days they were also tested in this same manner except that instead of being placed on the rotator and tested immediately, they were hooded, immobilized and allowed to

TABLE 1.—*Duration of Nystagmus of Pigeons with Rotation Experience on "Excited" and "Unexcited" Tests*

Bird No.	Excited	Unexcited	Excited	Unexcited	Excited	Unexcited	Excited	Unexcited	Excited	Unexcited	Excited, Average	Unexcited, Average
29.....	8.7	5.6	9.1	3.7	7.5	4.0	6.0	5.6	8.4	3.6	7.94	4.50
37.....	7.1	5.5	9.2	7.0	9.4	3.4	7.6	4.9	7.0	3.5	8.06	4.86
64.....	9.5	9.4	10.9	8.7	9.6	9.0	10.6	9.0	7.8	5.9	9.68	8.40
83.....	7.7	5.6	7.6	6.6	6.8	5.9	7.1	4.7	5.3	6.0	6.90	5.76
95.....	11.0	7.5	12.1	9.2	11.1	10.3	9.1	10.4	10.3	11.7	10.72	9.82
145.....	6.3	4.4	6.7	6.0	7.0	3.9	6.5	4.3	4.5	5.2	6.20	4.76
148.....	9.8	11.9	9.6	13.0	9.3	7.3	8.2	7.8	7.8	8.1	8.94	9.62
149.....	10.6	7.4	13.2	6.1	13.9	4.5	9.6	9.9	13.3	6.0	12.12	6.78
155.....	9.4	5.5	9.0	5.6	9.3	7.0	9.2	7.6	6.9	5.6	8.76	6.26
165.....	12.1	7.4	11.3	5.1	7.6	5.4	7.9	6.6	9.4	6.7	9.66	6.24
Average	9.22	7.02	9.87	7.10	9.15	6.07	8.18	7.08	8.07	6.23	8.898	6.700

remain unmolested on the rotator for twenty minutes prior to rotation. The duration of nystagmus for each bird on the successive "excited" and "unexcited" tests and the averages for all ten birds on the successive tests are given in the first ten columns of table 1. The average duration of nystagmus for each bird on the five "excited" and five "unexcited" tests are likewise given in the two columns at the extreme right of the table, marked "excited average" and "unexcited average." The values at the bottom of these last two columns give, respectively, the total average duration of nystagmus under the "excited" conditions of the experiment (8.898 seconds; probable error, $\pm$ 0.356) and the total average duration of nystagmus under the "unexcited" conditions (6.700 seconds; probable error, $\pm$ 0.400). The difference between these two averages is thus 2.198 seconds, and the probable error of this difference is $\pm$0.542. Therefore, since this difference is more than four times its probable error, it is practically certain that the greater average duration of nystagmus on the "excited" tests in comparison with the average duration of nystagmus on the "unexcited" tests is statistically

reliable, i. e., is due to the difference in the procedure employed under the two sets of experimental conditions rather than merely to chance variables.

However, in order to establish still more definitely the validity of this relationship and to obtain further information regarding the precise causal factors involved, ten pigeons without previous rotation experience were subjected to exactly the same procedure as that just described. The results obtained, which even more strikingly reveal the effect indicated by the preceding observations, are given in table 2. Here the difference between the "excited" average for the entire group (18.632 seconds; probable error, ± 0.582) and the "unexcited" average (12.182 seconds; probable error, ± 0.725) is 6.446 seconds, with a probable error of

TABLE 2.—*Duration of Nystagmus of Pigeons Without Rotation Experience on "Excited" and "Unexcited" Tests*

Bird No.	Excited	Unexcited	Excited	Unexcited	Excited	Unexcited	Excited	Unexcited	Excited	Unexcited	Excited, Average	Unexcited, Average
87.....	18.3	7.8	16.9	6.5	16.0	6.1	17.4	10.1	16.2	7.7	16.96	7.64
182.....	18.3	13.8	17.7	8.8	23.9	9.9	17.9	7.4	15.9	8.0	18.74	9.58
196.....	23.4	19.9	19.6	8.8	19.7	15.5	20.6	8.3	18.0	15.6	20.26	13.62
198.....	25.7	7.4	20.7	10.1	18.8	7.1	19.6	6.0	29.8	6.6	22.92	7.44
204.....	20.9	13.3	18.0	10.5	20.3	16.7	13.3	7.2	18.9	7.7	18.28	11.08
205.....	22.9	18.7	20.2	17.9	20.8	19.3	18.0	19.5	16.8	15.3	19.74	18.14
232.....	33.4	22.2	15.1	6.0	17.8	10.3	10.9	11.1	8.8	10.6	17.20	12.04
235.....	15.1	14.2	13.1	12.3	14.3	12.7	13.4	10.2	11.8	9.4	13.54	11.76
239.....	19.3	17.0	24.3	23.7	27.5	15.8	21.7	16.6	20.3	12.8	22.62	17.18
241.....	17.1	17.3	15.3	10.4	18.2	7.8	17.5	17.7	12.2	13.7	16.06	13.38
Average	21.44	15.16	18.09	11.50	19.73	12.12	17.03	11.41	16.87	10.74	18.632	12.186

± 0.932, which again leaves no doubt as to the existence of a causal relationship between the condition of the subjects just prior to rotation and the duration of the resulting nystagmus.

Although the data presented in tables 1 and 2 clearly reveal the source of the respective discrepancies noted between the average duration of nystagmus on the preliminary and final tests and on the first and last days of the habituation period for the first group of birds described, the underlying phenomenon, namely, the fact that a "rest" just preceding rotation decreases the duration of the ensuing response, remains in itself unexplained.

The use of the so-called "nystagmus test"—which has attained its most elaborate development in the hands of Dr. Robert Bárány and his many followers—as a specific clinical index to the functional integrity of the vestibular apparatus of the inner ear is necessarily predicated by the assumption that, given a normal sensorimotor mechanism, standardized stimulation produces a relatively "invariable" response. In

reality, both clinical and experimental observations have repeatedly demonstrated that there are often pronounced differences in nystagmus from person to person as well as in the same person from time to time without there being tangible evidence of corresponding variations in either the vestibular receptors or the respective effector mechanisms to which these wide variations in response might reasonably be attributed. However, as pointed out in a previous paper,[4] no specific physiologic explanation for these conspicuous "individual" and "functional" variations has yet been established; and there is likewise comparatively slight knowledge of the particular circumstances and conditions in which such variations are most likely to appear.

Bach [5] was one of the first to call attention to the notable differences in the nystagmus resulting from a standardized stimulation in different persons. On the basis of results obtained from an examination of sixty persons with presumably normal labyrinths, he concluded that "much therefore depends on the subject, as one almost invariably finds a pronounced nystagmus in normal persons of cultured status (*gebildeten Stände*) and in sensitive, easily excited and nervous persons, while on the contrary [one finds] often no nystagmus at all or only a slight nystagmus in stupid, indifferent and phlegmatic personalities." In short, Bach regarded the duration of nystagmus as primarily a function of the subject's nervous temperament and as only secondarily conditioned by specifically vestibular factors.

Observations similar to those made by Bach have been reported by Ruppert.[6] This investigator classified 115 persons (men, women and children) as (1) normal, (2) slightly abnormal and (3) decidedly abnormal, from the point of view of nervous excitability (*Reizbarkeit*) as revealed by the common neurologic tests. He found that not only could nystagmus be elicited by a smaller number of turns in the "nervous" subjects but that the response was also more rapid in them than in the normal subjects.

On the assumption that "an abnormally quick fatigability (*Ermüdbarkeit*) is a major symptom of neurasthenia and other functional nervous disorders," Ruppert concluded, in the light of the foregoing observations, that the nystagmus following bodily rotation is to be regarded essentially as a "fatigue symptom." This conclusion, although obviously not tenable for reasons shortly to be considered, is nevertheless interesting in that it might appear to explain, or at least to be

4. Mowrer, O. H., and Casey, W. R.: A Note on the Relationship Between Nystagmus Duration and Thyroid Condition, Endocrinology **16**:431, 1932.

5. Bach, L.: Ueber künstlich erzeugten Nystagmus bei normalen Individen und bei Taubstummen, Beitrag zur Physiologie des Ohrlabyrinthes, Arch. f. Augenh. **30**:10, 1894.

6. Ruppert, J.: Untersuchungen über den Drehnystagmus, Zentralbl. f. inn. Med. **27**:465, 1906.

superficially consistent with, the fact previously noted, namely, that "rest" just prior to rotation tends to shorten the duration of the resulting nystagmus. That nystagmus is not positively correlated with fatigue, however, is clearly indicated by the fact—which was probably not known to Ruppert—that repeated elicitation of nystagmus by means of successive periods of bodily rotation, instead of increasing the duration of the response as would be expected on the basis of Ruppert's fatigue hypothesis, usually causes (at least in naive subjects) a decided reduction in the duration and vigor of the response.[7] Likewise, it is equally evident that fatigue cannot, on the other hand, be responsible for this diminution of nystagmus with repetition, as has been proposed by Hoshino,[8] since this type of modification often persists much longer (often for days or even weeks) than any kind of fatigue effect could possibly be supposed to last. In short, then, there is no apparent justification for assuming that fatigue is correlated to any significant degree either positively or negatively with the duration of postrotational nystagmus; and it is accordingly necessary to seek elsewhere for a satisfactory explanation for the respective effects of "rest" and "excitement" on the duration of this response.

An outstanding peculiarity of the vestibular reaction system is that a momentary physical stimulus (e. g., either a positive or a negative angular acceleration lasting for only a fraction of a second), when sufficiently isolated temporally from a comparable stimulus of opposite sign, usually elicits a nystagmus which lasts many times as long as the original stimulus. Numerous theories of a "peripheral" mechanism—inertia and momentum movements of the endolymph, distortion of sensory epithelia, etc.—have been evolved by various writers in attempting to account for this discrepancy between the duration of the physical stimulus and the resulting response; but such hypotheses have repeatedly shown themselves to be inadequate to account for all the observed facts, and have been generally discarded by investigators in favor of the theory that the persistence of nystagmus after the withdrawal of the external stimulus is dependent on the action of a central neural mechanism of some sort.[9]

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7. (*a*) Mowrer, O. H.: The Reduction of Vestibular Nystagmus by Means of Repeated Elicitation, J. Comp. Psychol., to be published. (*b*) Footnote 2.

8. Hoshino, T.: Vestibuläre Reflexbewegungen des Auges beim normalen Kaninchen, Acta oto-laryng. **4**:328, 1922.

9. (*a*) Bárány, R.: Weitere Untersuchungen, Monatschr. f. Ohrenh. **41**:477, 1907. (*b*) Fischer, M. H.: Die Regulationsjunktionen des menschlichen Labyrinthes und Zusammenhänge mit verwandten Funktionen, Ergebn. d. Physiol. **27**: 209, 1928. (*c*) Mowrer, O. H.: Inverse Nystagmus: Its Probable Cause and Consequences, J. Exper. Psychol., to be published. (*d*) Lorente de Nó, R.: Ausgewahlte Kapitel aus der vergleichenden Physiologie des Labyrinthes, Ergebn. d. Physiol. **32**:73, 1931.

If the discrepancy between the duration of the physical stimulus and the resulting nystagmus is indeed due to the action of a "central" mechanism, it would be highly plausible to suppose that the duration of nystagmus might be easily influenced to a significant extent by general neural conditions. It will be recalled that both Bach and Ruppert found more conspicuous postrotational nystagmus in "nervous" than in normal subjects. Bárány,[9a] Wintermute,[10] Güttich [11] and others have reported generally augmented nystagmus in persons with neurasthenia [12] and other psychoneurotic types. Claude, Baruk and Aubry [13] found that nystagmus was either markedly diminished or entirely absent in a group of catatonic patients with dementia praecox, but that there was no noticeable departure in this respect in other types of insanity [14] from what might be expected in normal persons. Fenton [15] reported that "psychic conditions," such as worry, fear and excitement, may influence nystagmus, but he did not indicate the direction in which these factors may be expected to operate. Griffith [15a] reported the observation that muscular exertion immediately prior to bodily rotation tends to augment the postrotational nystagmus. Thus, to quote:

> Some twenty times during the series of investigations subjects came to the laboratory suffering from organic disturbances in no way connected with the experiments themselves. Occasionally a subject came in from violent exercise or from exertion on excessively warm days. In all these cases, the time of nystagmus, the number of eye-movements, and the time of the apparent movement of the visual field were appreciably increased. . . . From these figures, the effect of the organic disturbance is apparent. Furthermore, the figures show that the effect is only temporary . . .

Although the evidence just cited is not entirely unequivocal, it appears, in general, to support the hypothesis that the duration of post-

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10. Wintermute, G. P.: The Tests for Involvement of the Labyrinth in Suppurative Middle Ear Processes, California State J. Med. **7**:97, 1909.

11. Güttich, A.: Beitrag zur Physiologie des Vestibularapparates, Beitr. z. Anat., Physiol., Pathol. u. Therap. **7**:1, 1913.

12. Walker (Lancet **2**:1078, 1927) studied the nystagmus of five persons with neurasthenia, one of whom showed a "rather sluggish" nystagmus and the other four a "normal" nystagmus. However, the discrepancy between these observations and those of the writers cited may be due to the fact that the latter used rotation as a stimulus whereas Walker used caloric stimulation.

13. Claude, P. H.; Baruk, H., and Aubry, M.: Contribution à l'étude de la démence précoce catatonique: inexcitabilité labyrinthique au cours de la catatonie, Rev. neurol. **1**:976, 1927.

14. The vestibular responses of epileptic persons have been repeatedly investigated but with no very consistent results (Rabinowitsch, J. S.: Monatschr. f. Psychiat. u. Neurol. **70**:82, 1928).

15. Fenton, R. A.: Otolaryngologic Aspects of Commercial Aviation, Ann. Otol., Rhin. & Laryng. **40**:1070, 1931.

15a. Griffith, C. R.: The Organic Effects of Repeated Bodily Rotation, J. Exper. Psychol. **3**:15, 1920.

rotational nystagmus may be influenced to an appreciable degree by some condition in the central nervous system which may be conveniently, if perhaps somewhat arbitrarily, designated simply as "excitement." Moreover, this is the only theory which satisfactorily accounts for the fact that rotation which is preceded by a "rest" period so consistently elicits a nystagmus of shorter duration than does rotation which is not preceded by such a period.

Ross and Fearing [16] found that hooding and immobilizing pigeons (by the same method as previously described) cause a significant drop in body temperature; and Ivy [17] showed that, at least in decerebrate pigeons, temperature is an important factor in determining the duration of this response. It might be suggested, therefore, that the relatively shorter duration of nystagmus following bodily rotation which is preceded by a "rest" period is due to a drop in the birds' temperature. What appears more probable, however, and more in accord with the corroborative evidence reviewed in the preceding pages is that both the lowered temperature and the diminished nystagmus are common results of a lowered rate of living (reduced "excitement"), which is reflected both by the temperature-regulating mechanism and by the sensitivity of the nonacoustic labyrinth.

It has been incidentally mentioned in the preceding pages that repeated elicitation of nystagmus often leads to a substantial and relatively enduring reduction in the duration of this response. Since "excitement" has been shown to have a significant positive influence on the duration of nystagmus, it might reasonably be assumed—as has indeed been done by Güttich [11]—that the diminution of this response which usually occurs with repetition is therefore merely the result of a progressive reduction in "excitement," occasioned by the subject's gradual adjustment to the novelty and strangeness of the experimental or clinical situation. However, that such an assumption is not justified is indicated by the fact that, given certain special experimental conditions, repeated elicitation of nystagmus does not result in a reduction of the response [7a] and by the fact, also observed by me, that the mere handling and "gentling" of animal subjects is likewise ineffective from the point of view of modification of the nystagmus. Although it is not yet entirely clear what the mechanism is which is involved in the "habituation" of nystagmus, there is at present no reason to assume that it is at all intimately related to the phenomenon which has comprised the major thesis of the present paper.

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16. Ross, G., and Fearing, F.: Factors Influencing Temperature in Pigeons, with Special Reference to Level of Activity, Anaesthesia, and Diurnal Rhythm, J. Comp. Psychol., to be published.

17. Ivy, A. C.: Experimental Studies on the Brain Stem: II. Comparative Study of the Relation of the Cerebral Cortex to Vestibular Nystagmus, J. Comp. Neurol. **31**:1, 1919.

SUMMARY

The experimental results of the present study have demonstrated that, at least in the type of subject here used (pigeons), bodily rotation which is preceded by a "rest" period (during which the birds are immobilized and hooded) usually elicits a nystagmus of significantly shorter duration than does rotation which is not preceded by such a period. This finding might appear superficially to support the contention that postrotational nystagmus is a "fatigue symptom"; but certain considerations are advanced which make such an interpretation quite untenable. Evidence is cited which strongly suggests that the duration of nystagmus is conditioned by a "central" mechanism and that the conspicuous variations in this response frequently observed between different subjects and in the same subject on different occasions (and which cannot be accounted for by specifically vestibular factors) can be most adequately explained on the assumption that the functioning of this "central" mechanism may be strongly influenced by characteristic differences in nervous disposition as well as by temporarily prevailing neural conditions. It is believed, moreover, that the effect of "rest" just prior to rotation on the duration of the postrotational nystagmus is due to a temporary reduction of general nervous "excitement," which is reflected by a diminished reactivity of the "central" mechanism just mentioned. Finally, it is suggested that the so-called habituation of nystagmus is probably dependent neither on a reduction in "excitement," through the gradual adaptation of the subject to the testing situation, nor on any sort of fatigue effect, but rather, that it is dependent on a type of neural process which has been discussed in a paper cited at the beginning of the present report.

EFFECTS OF DRUGS ON CILIARY ACTIVITY OF MUCOSA OF UPPER RESPIRATORY TRACT

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Recent literature relating to the normal activity of the cilia of the mucosa of the upper respiratory tract has been plentiful, but there have been only a few scattered references to the effects of drugs. It was with this thought in mind that the present work was undertaken. The immediate direct effect of the various drugs used daily in the nose on the speed and continuation of ciliary activity seemed both interesting and clinically important.

As Yates,[1] Hilding [2] and Lucas [3] have pointed out, the activity of the cilia is an important physiologic factor in the defense mechanism of the upper respiratory tract. It is evident that any agent which interferes with the normal activity is detrimental to this defense mechanism.

Maxwell [4] studied the effects of various inorganic salts on ciliated epithelium; Seo,[5] the effects of changes in acidity of Ringer's solution and of changes in temperature. The reactions to certain calcium-precipitating substances were investigated by Wenner,[6] and the reac-

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1. Yates, A. L.: Methods of Estimating Activity of Ciliary Epithelium Within the Sinuses, J. Laryng. & Otol. **39**:554 (Oct.) 1924.

2. Hilding, Anderson: Ciliary Activity and Course of Secretion Currents of Nose, Proc. Staff Meet., Mayo Clin. **6**:285 (May) 1931; Influence of Ciliary Activity on Bacteriology of Nose: Direction of Ciliary Currents in Frontal Sinus of Dog, ibid. **6**:320 (May) 1931; Physiology of Drainage of Nasal Mucus: Flow of Mucus Currents Through Drainage System of Nasal Mucosa and Its Relation to Ciliary Activity, Arch. Otolaryng. **15**:92 (Jan.) 1932; Physiology of Drainage of Nasal Mucus: Cilia and Mucin in Mechanical Defense of Nasal Mucosa, Ann. Otol., Rhin. & Laryng. **41**:52 (March) 1932.

3. Lucas, A. M.: The Nasal Cavity and Direction of Fluid by Ciliary Movement in Macacus Rhesus, Am. J. Anat. **50**:141 (March) 1932.

4. Maxwell, S. S.: Effect of Salt Solutions on Ciliary Activity, Am. J. Physiol. **13**:154 (March) 1905.

5. Seo, Aizaburo: Ueber die Säurelähmung der Flimmerbewegungen, Jap. J. M. Sc. Tr., III, Biophysics **2**:257 (March) 1932; Ueber den Einfluss der Temperatur auf die Bewegung der Wimperkörperchen, ibid. **2**:189 (March) 1932.

6. Wenner, W. F.: Effect of Calcium Precipitating Substances on the Ciliated Epithelium of the Maxillary Sinus, J. Lab. & Clin. Med. **16**:341 (Jan.) 1931. Wenner, W. F., and Nemours, P. R.: The Influence of Calcium Combining Substances on the Maxillary Sinus Mucosa Under Various Conditions, Tr. Am. Laryng., Rhin. & Otol. Soc. **37**:170, 1931.

tions to various narcotics, by Nomura.[7] A study of the effects of ions on ciliary movements was made by Gray.[8]

N. Fox, after working with liquid petrolatum, menthol, eucalyptol and camphor in 1927[9] and 1930[10] reported no shrinking effect but definite destructive changes in the nasal mucosa of rabbits. These changes were most marked when 5 per cent menthol was used. Histologic study showed some deleterious effects produced by 1 per cent menthol, 5 per cent eucalyptol, 5 per cent camphor and liquid petrolatum. Again, in 1931,[11] he made similar histologic examinations after using ephedrine hydrochloride in a dilution of 1 : 50 and epinephrine hydrochloride in a dilution of 1 : 1,000. The results showed little if any injury after ephedrine, but definite destructive changes after epinephrine.

Stark[12] found inflammatory changes and scarcity of cilia after irrigations with a 2 per cent solution of sodium chloride and similar effects, though to a less extent, after tap water and 0.9 per cent sodium chloride were used. In 1932,[13] he reported that inflammatory changes were present in the nasal mucous membranes of three of ten rabbits treated with a spray of liquid petrolatum; in two of ten treated with ephedrine in oil, and in one of ten treated with ephedrine in water.

The following experimental work was done in an attempt to evaluate the effects of various drugs on the activity of the unprotected cilia of the mucosa of the upper respiratory tract of man, the dog and the guinea-pig:

1. A small series of determinations was made of the effects of certain drugs on the ciliary activity of strips of tissue mounted in a special microscope chamber.

2. Twenty determinations were made of the effects of a larger number of drugs on the ciliary activity of the unbroken mucosa of the turbinates of freshly killed guinea-pigs in situ.

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7. Nomura, S.: Influence of Narcotics on Ciliary Movements of the Gill of the Oyster, Proc. Soc. Exper. Biol. & Med. **25**:252, 1928.

8. Gray, J.: The Effect of Ions upon Ciliary Movement, Quart. J. Microscop. Sc. **64**:345 (March) 1920.

9. Fox, N.: Effect of Camphor, Eucalyptol and Menthol on Vascular State of Mucous Membrane, Arch. Otolaryng. **6**:112 (Aug.) 1927.

10. Fox, N.: Effect of Camphor, Eucalyptol and Menthol on Nasal Mucosa, Arch. Otolaryng. **11**:48 (Jan.) 1930.

11. Fox, N.: Chronic Effect of Epinephrine and Ephedrine on Nasal Mucosa, Arch. Otolaryng. **13**:73 (Jan.) 1931.

12. Stark, W. B.: Irrigations with Aqueous Solution; Their Effect on the Membranes of the Upper Respiratory Tract of the Rabbit, Arch. Otolaryng. **8**:47 (July) 1928.

13. Stark, W. B.: Effect of Mineral Oil and Solutions of Ephedrine on the Normal Nasal Mucous Membrane of the Rabbit, Arch. Otolaryng. **16**:39 (July) 1932.

DETERMINATIONS MADE ON EXCISED TISSUES

The first observations were made on excised tissue suspended in a circulating liquid in a special chamber placed under the microscope. It was found that Locke's mammalian buffered solution was best suited to prolonged ciliary activity. One piece of mucosa from a human frontal sinus which was placed in this solution at room temperature and changed to fresh solution every twenty-four hours maintained excellent general activity for seventy-seven hours after its removal from the patient. Some activity was noted even at the end of one hundred and twenty-one hours. This activity was surprising, as the tissue showed some thickening and was removed from a boy 14 years old who had an area of osteomyelitis of the frontal bone connecting with the frontal sinus.

Human tissue obtained from the operating room was used for the most part in these observations. Two series of experiments were made on tissue taken from a dog's nasopharynx. These showed the same response as the experiments on human tissue. All tissue was put in Locke's solution immediately on removal.

The apparatus used (fig. 1) varied only in minor details from that described by Lucas [14] in his work on Modiolus demissus. A Y tube, with suitable shut-off cocks, connected with a 10 cc. syringe without a plunger, was introduced between the supply of Locke's solution and the microscope chamber so that the various drugs could be introduced at will. The capacity of the Y tube and chamber was 1.5 cc. The chamber was placed on a thermostatically controlled warming stage which was kept at 98 F. In addition, all solutions were warmed before they were used in order to approximate body temperature as nearly as possible. The microscope was fitted with a 10 × objective and a binocular attachment with 15 × eyepieces. Preliminary determinations showed immediate marked slowing of all cilia on the introduction of either distilled or tap water. The activity returned to normal when Locke's solution again was permitted to flow over the cilia. This eliminated the use of a large number of drugs, because if aqueous solutions were used, it would not be known whether the results were due to the action of the drug or to the water solvent. The following drugs were selected for this part of the experiment, and were put in Locke's solution: 3 per cent ephedrine hydrochloride; 5 per cent cocaine hydrochloride; 1 per cent menthol; 1 per cent eucalyptol; 1 per cent thymol, and 1 per cent atropine sulphate. Determinations were made also with 0.5 per cent solutions of menthol, eucalyptol and thymol. Their action was the same as that of the 1 per cent solutions, except that a few minutes more were necessary to obtain the same reaction.

Very thin strips were cut from the tissue with a sharp razor blade and mounted in the examining chamber under the microscope. The flow of Locke's solution through the chamber was started immediately and was interrupted only during those periods when the action of the drugs was being observed. It was found that the flow could be stopped for at least forty-five minutes without causing any visible slowing of the cilia. The period of interruption for the determinations of

14. Lucas, A. M.: Methods of Study and the Relation of Ciliary Coordination to Ciliary Inhibition, Modiolus Demissus, J. Morphol. **53**:243 (June 5) 1932.

the effects of the drugs did not last longer than four or five minutes. Eight cubic centimeters of the drug solution to be tested was introduced through the syringe and Y tube. The Locke's solution in the chamber was allowed to be displaced by 3 cc. of the drug solution before the stop-watch was started. The period of observation was five minutes unless a definite reaction was noted sooner. The syringe, the Y tube and the tissue were washed thoroughly with Locke's solution after each determination.

The normal speed of the cilia of the mucosa of the respiratory tract of man, the dog and the guinea-pig is too slow for accurate measurement with the stroboscope because of the intense flicker. The following observations are based only on the gross changes in speed which could be detected definitely by the eye. Minor or questionable changes were

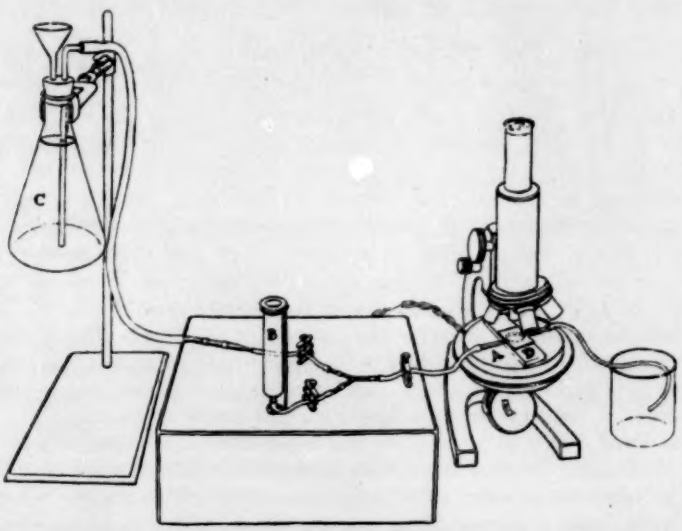


Fig. 1.—Apparatus used for the first series of determinations: *A*, the thermostatically controlled warming stage; *B*, the syringe barrel for solutions of the drugs; *C*, the flask containing Locke's solution; *D*, the sealed microscope chamber.

not recorded. Another variable factor which could not be eliminated was pointed out by Maxwell.[4] This is the fact that some cilia are more resistant than others and tend to maintain their speed after others have slowed down materially. The spontaneous cessation of ciliary activity noted by Gray [15] and Lucas [14] in their observations on marine animals was not seen in the ciliated epithelium of the upper respiratory tract, although certain areas often showed much slower activity than others. The reactions recorded in the tables are those shown by the majority of the cilia in the areas under observation.

15. Gray, J.: Mechanism of Ciliary Movement: Photographic and Stroboscopic Analysis, Proc. Roy. Soc. London, s.B **107**:313 (Dec.) 1930.

Table 1 shows the results of the small series of determinations made in the chamber.

Three per cent ephedrine hydrochloride produced no effect in eleven of the thirteen determinations. In the other two, it caused an increase in ciliary activity. In one of these two instances, the speed had been somewhat impaired by a preceding application of 1 per cent menthol.

Five per cent cocaine hydrochloride produced no effect in nine of the thirteen determinations. In the other four it caused slowing of ciliary activity. One per cent menthol caused a mild, but definite, slowing in all but two of the thirteen determinations. Recovery was always good.

One per cent thymol and 1 per cent eucalyptol produced slowing or cessation of ciliary activity in every determination. Recovery from the effects of either drug depended on instantaneous washing when a definite

TABLE 1.—*Results of Determinations Made in the Chamber* *

	Effect												
Drug	1	2	3	4	5	6	7	8	9	10	11	12	13
Ephedrine, 3%.....	0	0	0	0	0	0	0	0	0	I 2′	0	I	0
Cocaine, 5%.....	DR	DR	DR	0	0	0	0	0	0	0	DR	0	0
Menthol, 1%.....	DR 2′	DR	DR 2′	0	DR	DR	DR	DR	DR	DR	0	DR	DR 3′
Thymol, 1%.....	SR 1′30″	S 1′30″	DR 40″	DR 30″	DR 30″	DR 40″	DR 30″	S 3′15″	S 3′	S 3′	S 1′15″	S 1′	S 1′15″
Eucalyptol, 1%.....	SR 2′	S	S	S	S 2′	DR	S 3′	S 3′	S	S 3′	S 3′	S 2′15″	S 2′30″
Atropine, 1%.....	DR	0	0	0	0	0	0	0	0	0	0	0	0

* Time five minutes unless otherwise specified. I indicates increase; D, decrease; 0, no change; S, stopped; R, recovery after washing with Locke's solution.

reaction was noted. One per cent atropine sulphate produced no effect in twelve of the thirteen determinations.

DETERMINATIONS ON INTACT MUCOSA OF TURBINATE

Because of the possibility that some of the results obtained in the chamber were due to the action of the drug on the submucosa, a second series of determinations was made on an unbroken surface of mucous membrane. For this series, the turbinate of a freshly killed guinea-pig was exposed by removing the nasal bone and part of the superior maxilla. Care was taken not to break the mucous membrane covering of the turbinate itself.

A microscope from which the stage had been removed was placed in a horizontal position (fig. 2). A specially designed tray was adjusted across its frame so that when the guinea-pig was placed in the tray the exposed turbinate could be brought into focus. Illumination was by means of a bright beam of light thrown on the turbinate by a concave mirror, located beside the barrel of the microscope. The illumination was enhanced by a small piece of tinfoil, placed

behind and below the turbinate, so that it acted as a reflector. This produced areas of bright light reflexes on the turbinate, which made visible the motion imparted to the surface of the mucous sheet by the activity of the cilia immersed within it. The observations were made at room temperature.

A flask of Locke's solution was held by a standard and connected by tubing to a glass nozzle which was so situated that a stream of the solution could be made to flow on the exposed turbinate at will. Here again, a small, steady stream was used, except during the time when the drug was being applied and observations were being made. After each application, the turbinate was washed carefully with Locke's solution. Observations were made through the microscope from the moment the drug came in contact with the mucosa. Care was taken to allow the drug to flow over the turbinate often enough to prevent drying. This was extremely important, for the exposed surface of the turbinate tended to dry very quickly, and even the minimum of drying caused a decrease or cessation of ciliary

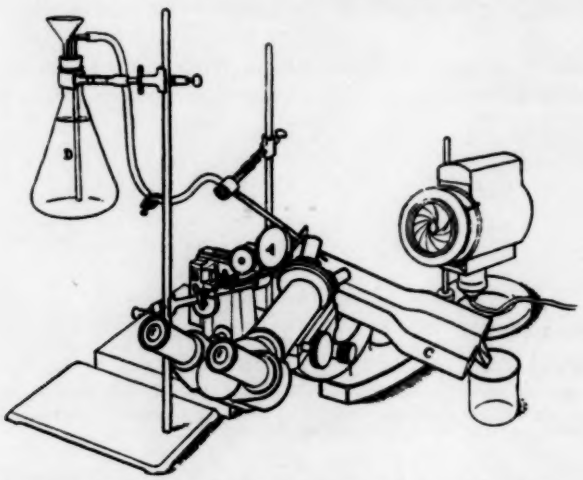


Fig. 2.—Apparatus used for the second series of determinations: *A*, the concave mirror to concentrate light beams on the turbinate; *B*, the mechanism for horizontal and vertical adjustment of *C*, the tray for the guinea-pig; *D*, the flask containing Locke's solution.

activity. Unless the drying was marked, the cilia returned to normal activity again when properly moistened.

Continued washing with Locke's solution naturally resulted in the depletion or removal of the protective mucous sheet which is normally present. This was not detrimental to the results, however, because the observations were made on the action of the drugs on the cilia themselves. The depletion of the mucous sheet merely allowed the drugs to exert their action more easily and directly on the cilia. Prolonged washing with the Locke's solution did not impair the activity of the cilia in any way. Tap or distilled water, however, caused marked slowing or cessation of ciliary activity in a variable length of time. When the water was used on a fresh turbinate, with the mucous sheet intact, as

much as twenty minutes elapsed before this effect was noticed. On the other hand, after the mucous sheet had been impaired by previous washing, the effect appeared in about three minutes.

Drugs which are most commonly used in the treatment of the nose were selected. Locke's solution was used as a solvent for all drugs which were compatible with it. The others were placed in an aqueous solution because of their marked tendency to form a precipitate with Locke's solution. Table 2 shows the results obtained with those drugs which were placed in Locke's solution.

Five per cent cocaine hydrochloride produced no appreciable effect in eight of the ten determinations but a definite slowing of ciliary activity in two. Ten per cent cocaine hydrochloride impaired the speed of the cilia in all but one instance. Twenty per cent cocaine hydrochloride produced a more immediate slowing or cessation of activity in every determination.

Three per cent ephedrine hydrochloride produced a very slight sustained speeding of ciliary activity in eleven of the twenty determinations. In most of these cases the activity of the cilia was below par before the ephedrine was applied because of poor recovery from a previous application of a drug. In the other nine determinations it had no appreciable effect.

Five-tenths per cent menthol produced a decrease in ciliary activity in all but two of the twenty determinations, and 1 per cent menthol, in all but one. The reaction to the 0.5 per cent solution was milder and took place more slowly. Recovery was always good.

Five-tenths and 1 per cent thymol invariably produced a marked decrease or cessation of ciliary activity. If the drug was allowed to act until the cilia stopped beating, there was no recovery.

Five-tenths per cent eucalyptol had no apparent effect on the ciliary activity, except in one instance, when its application resulted in a definite sustained increase. One per cent eucalyptol produced a decrease or cessation of activity in every test. Recovery was usually fair, unless the cilia had stopped beating. When that occurred there was no recovery.

In some instances an initial speeding was noted with menthol, eucalyptol and thymol. This was not constant or definite enough to merit recording, however.

Table 3 shows the results obtained with the drugs used in aqueous solution. The interpretation of this table is modified by the slowing factor produced by the water solvent. It will be noted that there are ten determinations each on 5, 10 and 20 per cent cocaine in Locke's solution in table 2 and ten each on the same concentrations of cocaine in aqueous solution in table 3. This was done in order to link the two tables and simplify the interpretation of table 3. In table 2, the 5 per cent cocaine in Locke's solution caused a decrease in ciliary activity in only two of

TABLE 2.—*Results Obtained on Intact Turbinate with Drugs in Locke's Solution* *

Drug	Effect																			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Cocaine, 5%.....	0	DR	0	0	0 3′	0 3′	0	0	DR 4′	0 3′										
Cocaine, 10%..... ..	DR 3′15″	DR 2′15″	DRa 1′12″	DR 4′35″	0	DR 3′35″	DR 2′	DR 2′	DR 1′30″	DR 2′										
Cocaine, 20%.....	SR 2′	SR 1′30″	DRa 35″	DR 1′30″	SR 45″	SR 30″	SR 30″	DR 2′	DR 1′	DR 1′										
Ephedrine, 3%.....	Ia	Ia	Ia	0	0	0 3′	0 3′	I	I	I	0 3′	Ia	0	0	I 45″	0 1′45″	Ia 1′30″	Ia 1′50″	Ia 1′20″	0 4′
Menthol, 1%.....	DR 2′30″	DR 4′	DR	0	DR 3′	DR 2′15″	DR 3′	DR 4′10″	DR 1′15″	DR 2′30″	DR	DR	DR	DR 3′30″	DR 3′10″	DR	DR 4′	DR	DR 3′45″	DR
Menthol, 0.5%..... ..	0	0	DR	DR 3′	DR 4′	DR	DR 2′30″	DR 3′	DR 4′	DR	DR 3′	DR 1′	DR	DR 2′	DR	DR 3′15″	DR 3′15″	DR 3′15″	DR 2′30″	DR
Thymol, 0.5%.....	S 1′	DR 2′	S 2′	S 1′	DR 1′15″	S 1′20″	S 1′10″	S 1′15″	S 2′	DR 1′30″	DR 30″	DR 49′	DR 1′30″	DR 30″	S 45′	S 1′	S 1′20″	DR 40″	DR 1′15″	DR 1′
Thymol, 1%..... ..	S 1′	S 2′	S 2′	DR 30″	S 1′	DR 50″	S 1′30″	DR 30″	DR 45″	S 45″	DR 45″	DR 1′	S 1′10″	DR 1′30″	DR 30″	DR 30″	DR 30″	DR 1′	DR 15″	DR 30″
Eucalyptol, 0.5%.....	0	0	0	0	0	0	0	0	0	0	0	0	0	0	I	0	0	0	0	0
Eucalyptol, 1%.....	DR 4′20″	DR 3′51″	S 2′	DR 3′	DR 3′	DR	DR 1′30″	DR 1′45″	DR 3′30″	DR 1′15″	DR 3′30″	DR 3′	DR 1′	DR 2′	S 4′5″	DR 4′	DR 2′	DR 1′	DR 1′20″	DR 2′5″

* Time five minutes, unless otherwise specified. I indicates increase; D, decrease; 0, no change; a, activity impaired before application of drug; S, stopped; R, recovery after washing with Locke's solution.

TABLE 3.—*Results Obtained on Intact Turbinate with Drugs in Aqueous Solution* *

Drug	Effect																			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Cocaine, 5%.....	DR 3′30″	DR 4′	DR 1′	DR 2′	DR 1′15″	DR 4′15″	DR 2′40″	DR 1′50″	DR 1′5″	DR 2′3″										
Cocaine, 10%.....	DR	DR 3′	SR 3′	DR 3′	DR 1′15″	DR	DR 3′30″	DR 1′	DR	DR 2′40″										
Cocaine, 20%.....	DR 3′	DR	SR 0′	DR 1′	DR 3′	DR 1′40″	DR 1′50″	DR 1′	DR 3′	DR 4′	DR 1′	DR 2′								
Epinephrine, 1:1,000....	S 45″	SR 0′	S 0′	S 1′	DR 20″	S 55″	DR 28″	SR 45″	SR 1′30″	S 30″	SR 0′	SR 0′	DR 5″	DR 0′	DR 0′	DR 5″	DR 0′	DR 5″	DR 10″	S 1′
Mercurochrome, 2%....	DR 1′	DR 1′	S 1′	DR 1′	DR 1′	S 1′	S 1′	DR 2′	DR 30″	DR 20″	DR 20″	S 2′30″	DR 2′30″	S 2′	S 1′40″	DR 1′	S 2′	DR 1′30″	DR 2′7″	DR
Silver nitrate, 0.5%.....	S 1′	S 0′	S 1′15″	S 1′	S 0′	S 15″	S 0′	S 1′	S 30″	S 1′30″	S 0′	S 0′	S 15″	S 0′	S 0′	S 0′	S 0′	S 0′	S 0′	S 0′
Zinc sulphate, 2%.....	SR 30″	S 2″	SR 3′	SR 2′	SR 15″	DR 55″	SR 2″	DR 15″	DR 15″	DR 15″	DR 10″	DR 15″	DR 30″	DR 30″	DR 1′30″	DR 1′	DR 1′	S 1′	DR 38″	DR 30″
Mild silver protein, 5%.	†DR	†SR	†DR 4′	DR 2′45″	†DR	†DR 4′	†DR 3′30″	†DR 2′	†DR 3′	†DR	†DR 3′20″	†DR	†DR	†SR 2′15″	†DR 2′	†DR	†DR 3′10″	†DR 4′	†DR	†DR
Mild silver protein, 10%	†DR	†DR 4′	†DR	†DR	†DR	†DR 3′30″	†DR 4′30″	†DR 2′	†DR 2′	†DR 3′30″	†DR 3′	†DR 2′	†DR 2′30″	†DR	†DR 2′	†DR 3′40″	†DR 3′	†DR 4′45″	I	†DR
Mild silver protein, 20%	†S	†DR 2′30″	†DR 4′	†DR	†DR 3′	†DR 2′45″	†DR 1′15″	†DR 2′30″	†S	†DR 3′15″	†DR 4′	†DR 1′30″	†DR 4′	†DR 3′10″	I	†DR 3′30″	†DR 4′30″	†DR 4′	†DR	I

* Time five minutes, unless otherwise specified. I indicates increase; D, decrease; 0, no change; S, stopped; R, recovery.
† Initial speeding

the ten determinations. In table 3, 5 per cent cocaine in water caused a slowing in each of the ten determinations. The action of 10 and of 20 per cent cocaine was about the same in each solvent. This would seem to indicate that in the 5 per cent concentration the drug had little or no effect, the action shown in table 3 being due mainly to the water solvent. In the higher concentrations the cocaine itself produced the slowing. This was more marked and appeared sooner after application of the drug in the 20 per cent concentration. Recovery was uniformly good, even after the cilia had ceased beating.

Epinephrine hydrochloride in a concentration of 1:1,000 caused almost immediate slowing or stopping. In all but six of the twenty determinations recovery was good.

The interpretation of the remainder of table 3 is complicated by the fact that mercurochrome, silver nitrate, zinc sulphate and mild silver protein produce a precipitate when they come in contact with the tissues or with the Locke's solution used to wash off the drugs. Plain water was tried as a wash, but this resulted in too many instances in complete cessation of ciliary activity. In these instances the activity could not be recovered.

The reactions to 2 per cent mercurochrome, 0.5 per cent silver nitrate and 2 per cent zinc sulphate were definite and prompt. They were too marked and were noted too quickly to be due to the water solvent. They were too definite and prompt to be obscured by the formation of the precipitate. Cessation or marked decrease of activity was produced quickly by 2 per cent mercurochrome. If the drug was washed off during the stage of decreased activity, the recovery was uniformly good. Five-tenths per cent silver nitrate produced immediate cessation of activity, from which there was no recovery. Two per cent zinc sulphate produced immediate slowing or cessation of ciliary activity from which there was usually a fairly good recovery.

Mild silver protein in concentrations of 5, 10 and 20 per cent invariably produced an initial speeding, which usually lasted for two, three or even four minutes. In one observation with 10 per cent mild silver protein and in two with a 20 per cent solution the increased activity was maintained for the full five minutes of the observations. The initial speeding was followed by a slowing, which appeared coincidentally with a decreased visibility due to the formation of a precipitate. This slowing also appeared about the time that water would produce the same effect. Recovery was uniformly good, except in two of the determinations with 20 per cent mild silver protein. In these two, the ciliary beat stopped altogether and there was no recovery. In all instances, however, the precipitate, which remained on the surface after washing, interfered with the visibility of the ciliary activity. This seems to indi-

cate that the slowing was not due to the presence of the precipitate itself. It seems reasonable to assume that the water solvent was responsible for the slowing.

Some determinations were done on the turbinate of the living guinea-pig. These were too few to merit report at the present time. They were sufficient, however, to indicate that the effect of the drugs was the same on the live turbinate as on the turbinate of the freshly killed animal. An attempt was made to preserve the mucous sheet by reducing to the absolute minimum the amount of fluid applied. Working conditions were not so ideal nor so conducive to satisfactory observations as when the dead guinea-pig was used. This was due to the accumulation of blood clots and the dilution of the drug by serum and blood from the cut tissues.

CONCLUSIONS

1. Tap and distilled water when applied to the mucosa of the upper respiratory tract cause slowing of the ciliary beat.

2. Three per cent ephedrine hydrochloride is not detrimental to ciliary activity but at times increases it slightly.

3. Five per cent cocaine hydrochloride is not detrimental to ciliary activity, but 10 and 20 per cent solutions produce definite slowing, with good recovery.

4. Mild silver protein in concentrations of 5, 10 and 20 per cent produces an initial speeding of ciliary activity. This is followed by a slowing, which may be due to the water solvent rather than to the drug.

5. Five-tenths per cent eucalyptol has no deleterious effect on ciliary activity.

6. Five-tenths per cent menthol and, to a greater degree, 1 per cent menthol have a mildly depressing effect on ciliary activity.

7. One per cent thymol, 0.5 per cent thymol and 1 per cent eucalyptol, in the order named, are definitely detrimental to ciliary activity.

8. A 1 : 1,000 solution of epinephrine hydrochloride, 2 per cent zinc sulphate and 2 per cent mercurochrome, in the order named, are definitely detrimental to ciliary activity.

9. Five-tenths per cent silver nitrate is immediately and fatally detrimental to ciliary activity. In no instance was it possible to start the cilia beating again after its application.

Clinical Notes

A NEW SOFT PALATE RETRACTOR AND ADENOID PUNCH

ANDREW A. LOVE, M.D., LOS ANGELES

The instruments presented are a basket punch for removing the remnants of adenoids and soft palate retractors in sizes suitable for use in adults and children.

Otolaryngologists are familiar with the difficulty of removing the adenoid mass completely and with the necessity of minimizing trauma to the important structures

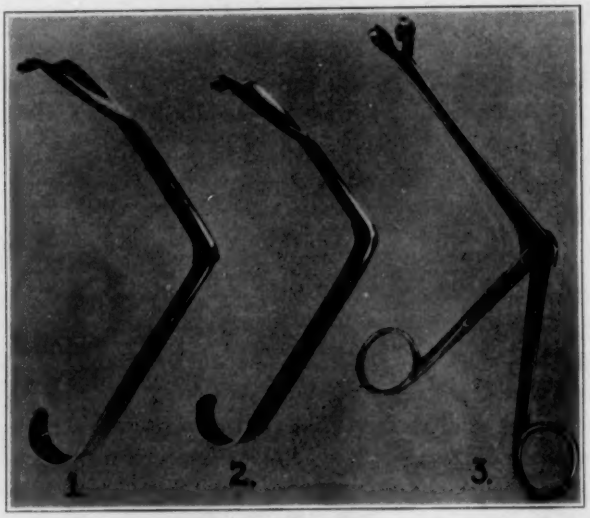


Fig. 1.—Instruments *1* and *2* are soft palate retractors in sizes suitable for adults and children. The handle is grasped with the left hand, so that the right hand is free. A tongue depressor that is held by an assistant is usually necessary. Instrument *3* is the basket punch used to remove the remnants of adenoids. The cups are made in such a way that it is practically impossible to lose a piece of tissue.

nearby. The fact that the epipharynx cannot be well visualized directly during or after operation accounts in a large part for the many incomplete operative attempts in this region. It was with this problem in mind that the instruments were devised.

The soft palate retractors give the greatest possible direct view of the epipharynx through the mouth, and at the same time afford ample space for surgical manipulation. They are useful for clinical examination, but are of greatest value, I believe,

in the inspection after removal of adenoids to determine whether the desired amount of tissue has been removed. Direct visualization is certainly more accurate than palpation with the index finger.

When remnants of the adenoids are present, they can be removed quite easily with the basket punch. The punch is also made for a universal handle.

Fig. 2.—An enlargement of the cupped end of the basket punch.

The soft palate retractor that I have described also aids greatly in the placing of a postnasal pack.

The instruments were made for me by V. Mueller & Company.

DERMOID CYST OF THE NASAL DORSUM

LYLE S. POWELL, M.D., LAWRENCE, KAN.

Recently Luongo[1] reported a case of dermoid cyst of the nasal dorsum in a woman, aged 30. His statement that there are only a few cases reported in the foreign literature and only one in the American literature prompts me to add the case of a child 15 months of age.

REPORT OF A CASE

History.—Patsy A., a full term child, weighed 7½ pounds (3.4 Kg.) at birth. At the time of examination the weight was 22 pounds (10 Kg.). She had not had any illnesses or irregularities. At about 3 months of age, the father, a physician, noted a swelling in the skin exactly in the midline over the nasion. This had gradually enlarged from the size of a small pea to that of a small marble. The father wished something done, as he feared a developmental deformity in the bridge of the nose from the pressure of the tumor.

Examination.—The patient was manifestly a healthy, robust child of about the stated age. Examination gave negative results except for a swelling in the skin

1. Luongo, R. A.: Dermoid Cyst of the Nasal Dorsum, Arch. Otolaryng. **17**:755 (June) 1933.

in the midline over the nasion. This swelling was ovoid and about 10 by 12 mm. in diameter. It was soft on palpation and fluctuated slightly. It was apparently movable. There was no redness or other signs of inflammation. Examination of the interior of the nose gave negative results. The father agreed that the tumor was very likely either a sebaceous cyst or a dermoid cyst, most probably the latter. Surgical removal was advised.

Operation.—With the patient under general anesthesia, a vertical linear incision was made through the skin, immediately disclosing an ovoid sac which seemed freely movable, but which actually was attached to the perichondrium at the nasion. It was exactly in the midline. This sac was carefully dissected out, and a bit of the perichondrium was removed with the sac at its basal attachment. The wound was closed without sutures, and uneventful convalescence followed with no scarring.

When the specimen was opened, it was found to be filled with a whitish, cheesy material and innumerable long red hairs.

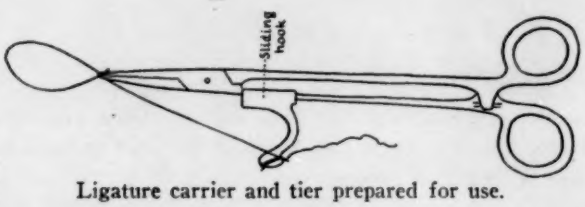
COMMENT

Undoubtedly these dermoid cysts occur as a result of sequestration of fragments of a surface epithelium during the fusion of the embryonal fissures, in this case most likely the orbitonasal fissure.

LIGATURE CARRIER AND TIER

M. L. Harris, M.D., Brooklyn

Bleeding in the tonsillar fossa after tonsillectomy at times is alarming and must be controlled with a ligature. Ligation of a bleeding vessel is the safest procedure, and quite a number of instruments have been devised to accomplish this.



Ligature carrier and tier prepared for use.

The instrument that I present is simple and facilitates an established procedure. It consists of a straight clamp with a sliding hook on one shank, to which one end of the ligature is attached.

To apply a ligature, the bleeding point is seized with a regular tonsillar artery clamp. A no. 1 catgut slip knot is made, and the fixed end is caught by the instrument close to the knot. The long end is slung about the sliding hook and tied. The loop is then slipped over the clamp, and with one hand manipulating this clamp (on the bleeding point) while the other hand holds the instrument to the clamped tissue, the loop is pulled tight by sliding the hook, as in manipulating the ordinary snare. The clamp and instrument are then removed, and the ligature is cut short.

USE OF A TAPERING TUBE SUTURED IN THE TRACHEA TO PREVENT ASPIRATION DURING AND AFTER LARYNGECTOMY

ROBERT STEELE IRVINE, M.D., SAN FRANCISCO

Utilizing the principle of lower-deck ventilation of ships, I successfully prevented aspiration by suturing a tapering tube in the trachea to prevent aspiration during and after laryngectomy in two cases, and, encouraged by the interest shown by several surgeons, I am passing on the idea. Briefly, the method is as follows:

The tapering end is cut from a Valentine irrigating tube. The larynx and upper part of the trachea are skeletonized, local anesthesia being used, and the moment the trachea is severed, the smaller end of the flexible tube is snugly fitted into it and secured by two heavy sutures, which are passed from the skin through the trachea and tube and tied over lead buttons. These sutures are not disturbed until healing is completed.

If desired, it is a simple matter at this point to connect this tube with the rigid tube of a standard gas apparatus and proceed with dissection and closure under general anesthesia. A small opening cut in the side acts as a satisfactory valve to regulate rebreathing.

After the operation is completed, the tube is cut off, but 2 or 3 inches (2.54 or 5.08 cm.) are left attached to the trachea and protruding above the dressings. This provides a satisfactory airway through which mucus and crusts may be removed by suction or forceps. A small wire-meshed household strainer tied about the neck protects against foreign bodies, and, when covered, serves as a warm air chamber.

The enlarged end of the Valentine irrigator is 12.5 cm. long and tapers from 1 cm. in diameter where it is cemented to the rubber tubing to 2.5 cm at its larger end. The tips may be secured separately from the tubing, if desired.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTIONAL TESTS OF HEARING

ROBERT SONNENSCHEIN, M.D.
AND
NORMAN LESHIN, M.D.
CHICAGO

As noted in the last review,[1] so again we observe that the number of papers published on functional testing of hearing is not very large. Furthermore, the articles in the American and the English literature far outnumber those in the German.

According to Fletcher,[2] the advice now given patients with impaired hearing regarding the use of hearing devices, namely, that they try two or three different sets and then determine for themselves which, if any, is the best for them, is far from satisfactory from the patient's point of view. The author presents some facts which have been determined in the research studies of the Bell Telephone Laboratories bearing on this subject which may aid otologists in enabling them to give their patients scientific advice on this subject.

The variations of the level of speech for a person with normal hearing cover a range of from 40 to 70 decibels above the threshold of hearing the faintest speech sounds, being about 10 decibels above the level of noise. A person having a uniform loss of hearing of 30 decibels will have no difficulty in interpreting conversational speech 1 meter away from the mouth of the speaker. A patient with a loss of hearing of 40 decibels has a borderline case of deafness, and in Fletcher's experience will not require the use of a hearing aid as the two people speaking will compensate easily for the loss of hearing. A patient with a loss of hearing of 50 decibels requires a set having an amplification of about 10 decibels. In general, a person having a loss of hearing L requires a set having an amplification of L-40. If a set were made with a varying amplification from 0 to 40, it would be satisfactory for persons with a deafness of from 40 to 80. The compromise between the dis-

1. Sonnenschein, Robert, and Leshin, Norman: Functional Tests of Hearing, Arch. Otolaryng. **17**:88 (Jan.) 1933.

2. Fletcher, Harry: Can We Scientifically Advise Patients as to the Effectiveness of Hearing Aids? Ann. Otol., Rhin. & Laryng. **41**:727 (Sept.) 1932.

tance of the speaker from the microphone and the amplification of the hearing aid must be made for the more severe types of deafness. These assertions are based on the following assumptions: first, that the loss of hearing is uniform; second, that the sound intensity involved is such that it will not hurt the ear, and third, that the amplification of the hearing aid is uniform. The first is seldom true; the second is true only with moderate degrees of deafness, and the third is true only in specially designed sets. To determine what recommendations to make concerning the use of hearing aids, the division of patients into the following four classifications may be useful (the loss of hearing applies mainly to the range of from 256 to 4,096 cycles in which region the recognition of the speech sounds are determined): The first class, with a loss of hearing of from 0 to 40 decibels, will not receive sufficient benefit from a hearing aid to justify the inconvenience of using it. The second group, with a loss of hearing of from 40 to 80 decibels, are candidates for hearing aids. The third class, with a loss of hearing of from 80 to 110 decibels, require more powerful aids which can be produced only by a vacuum tube equipment. At the present time, these sets are very bulky and expensive. The last group, with a loss of hearing from 110 decibels to total loss, cannot be helped by any equipment now available. To determine the effectiveness of any set for conversational purposes for persons whose loss of hearing is known, Fletcher has worked out a table by which the merit of a hearing device can be determined if the amplification characteristics of the instrument and the loss of hearing of the patient are known. This table is shown in the article. Fletcher suggests that a committee be appointed by the American Laryngological, Rhinological and Otological Society to obtain amplification curves for all the sets which are commercially available for the use of the members. In this way some may become sufficiently interested to try the procedure outlined by Fletcher.

Bunch [3] brings out the point that the audiometer should not be considered mainly a research instrument, as most of the work reported seems to show, but that it also is of considerable value in clinical work. Audiometric tests have been made as a routine measure in all cases of aural conditions for the past two years in the otological clinic of Washington University Medical School. The junior member of the clinical staff receives a course of instruction in the use of tuning forks, audiometers and other devices used in functional tests of hearing and in the interpretation of the results. After he has made a complete examination of the patient, a senior member of the clinical staff criticizes his observations and assists him in the diagnosis and in the

3. Bunch, C. C.: The Use of Various Types of Audiometers in Clinical Work, Ann. Otol., Rhin. & Laryng. **41**: 712 (Sept.) 1932.

proper method of treatment. This procedure results in a complete and careful examination in each case and, in addition, in the proper training of the junior member. Bunch also shows the variations in audiograms made under different conditions, namely, in a sound-proof room, in a fairly quiet place with a 2 *A* audiometer and in a sound-proof room with a 1 *A* and a 4 *A* audiometer on the same patient. All these audiograms showed variations, demonstrating the futility of comparing records with standards made under other conditions. In the work at the otological clinic at Washington University Medical School, a tentative normal for the conditions of the test is established and used as a standard. The examiner is instructed as to the significance of this normal, and his interpretations are based on it.

The audiometric tests give direct information regarding the progress of a condition which is undergoing treatment, as for instance, otitis media. The audiograms are of great value in following closely the efficacy of a specific treatment. Bunch also presents audiograms and case histories to bring out these various points. He also shows the difficulty encountered at times in making a diagnosis of aural conditions from the information given in the audiogram alone.

Guild [4] presents a method of classifying audiograms used as a basic part of the research program in the Otological Research Laboratory, Johns Hopkins University. The purpose is to facilitate the objective study of these records of hearing acuity from the standpoint of possible correlations with any of the other facts known about the patients, such as their medical history and age. This method is for audiograms obtained with the Western Electric 1 *A* type audiometer. The adaptation of this method for the 2 *A* audiometer is described. For those interested, we refer to the original article for a complete description.

Thaler [5] investigated the auditory acuity of 30 patients in a medical ward with a high cholesterol content of the blood. He wanted to determine whether the lipoidal infiltration of the auditory end-organ as reported by Fineberg in cases of hypercholesteremia resulted in a diminution of auditory acuity. All these patients showed an absolute loss of hearing ranging from 16 per cent in the second decade to 33 and 32 per cent in the fifth and sixth decades, respectively. These figures compared with those of Bunch in his study of loss of hearing in nonotologic patients in successive decades showed a greater loss, progressively increasing from 3 to 15 per cent. In a later paper Bunch reported a loss of hearing of 16 per cent in a group of 251 persons of all ages with syphilis, malignant growths, arteriosclerosis, cardiac dis-

4. Guild, S. R.: A Method of Classifying Audiograms, Laryngoscope **42**: 821 (Nov.) 1932.

5. Thaler, Nathan: Auditory Acuity in Hypercholesteremia, Laryngoscope **42**:741 (Oct.) 1932.

ease and hypertension. According to these reports, it is evident that systemic disease is not the etiologic factor in the production of marked loss of hearing. Thaler, in view of the constantly progressive impairment of hearing with increasing age found in this series of cases of systemic disease accompanied by hypercholesteremia, concludes that the progressive loss of hearing in this group is attributable to the hypocholesteremia with its attendant histologic changes.

Ciocco[6] reports the results of a statistical analysis of the hearing of 1,980 persons examined with a Western Electric 1 *A* audiometer, tuning forks, monochord, etc. These patients were selected at random from several medical and surgical wards with the cooperation of the staffs of the hospitals, no previous tests of hearing having been made. The audiograms were classified according to the method described by Guild, for research as well as for clinical purposes. Eliminating those persons in whom the responses were entirely unreliable and those for whom audiograms could not be classified, the total number was reduced to 1,847. According to the evidence found in this investigation, the 4,096 double vibration dip which Drury states is perhaps indicative of a syphilitic taint is more likely the first indication of a degenerative change in hearing, which may continue until it reaches that stage most common for advanced age, namely, a loss of acuity for all high tones. The findings regarding age and sex confirm those reported by Bunch and Raiford. With advancing age, there is a decrease in the perception of the upper tone limit, and apparently the normal limit for loss of acuity is for tones of 2,048 or 4,096 double vibrations. For the monochord, they found a lowering of the upper limit for air conduction by about 1,000 double vibrations for each decade above the second. They are inclined to favor the hypothesis that this impairment is due to an infection occurring some time in life, following which the hearing remains stationary until the end, as opposed to the belief that the loss is gradual with increasing age. The greater number of cases, therefore, involved with increasing age is probably due to the greater probability of a person contracting during a longer span of life the etiologic factor of the impairment. Males show a loss of hearing for tones of 4,096 double vibrations and above more frequently than females, probably because the latter lead a more sheltered life and are not exposed as much to injurious noises. On the other hand, it may simply be a true sexual difference. Ciocco also shows evidence indicating that inflammation of the middle ear may affect the acuity for high tones, which may be interpreted as meaning that the inner ear has been involved. Finally, the analyses made with

6. Ciocco, Antonio: Observations on the Hearing of 1,980 Individuals: A Biometric Study, Laryngoscope **42**:837 (Nov.) 1932.

respect to the possible influence of certain diseases, such as cardiovascular changes, syphilis, malignant tumors, gastro-intestinal diseases, tuberculosis and goiters, show that persons with these diseases reveal no greater lowering of acuity for high tones than those who are free from these conditions.

Fishbein [7] states that the majority of adults over 35 years of age have defective hearing at least in one ear. He also mentions that it is now known that deafness commences in early life and childhood, even though it may not be noticeable, at times, until middle age. The terms deaf, deafened and hard of hearing which are often misused and confused should be clarified. He defines them as follows: "Deaf—Those who are congenitally deaf and have no usable residual hearing. Deafened—Those who have no residual hearing, but who at one time had usable hearing. Hard of Hearing—Those with defective hearing but who still have usable residual hearing."

An ear can be considered normal when it can hear a watch tick at from 30 to 50 inches (76.2 to 127 cm.), a conversational voice from 60 to 70 feet (18 to 21 meters), whispered voice at 20 feet (6 meters), musical tones ranging between 18 and 41,000 double vibrations per second and a tuning fork with 128 double vibrations twice as long by air as by bone conduction. The following tests conducted in a quiet room should be performed to determine the acuity of hearing: the watch tick, whispered voice, conversational voice, tuning fork or audiometer, Galton whistle, Rinne, Weber, Politzer and Schwabach tests.

Records of 100 cases taken from the otologic clinic of the North End Dispensary showed 38 persons with a history of nasal discharge, aural discharge, nasal obstruction, tonsillitis or other conditions resulting from rhinopharyngeal pathologic processes. These figures were similar to those reported by Sturm, who found that 32 per cent of 200 cases of deafness were due to nasopharyngeal disease in childhood. He also stated that he rarely saw a perfectly normal drum in a child with adenoids. Fishbein suggests the following plan of procedure: a careful history; hearing tests, as mentioned previously; local examination of the tympanic membrane and eustachian tube; a general physical examination, and complete laboratory tests, such as examination of the urine and stool, determination of the blood chemistry, basal metabolism and Wassermann reaction, and also roentgenograms of the teeth, sinuses and mastoid processes.

Fishbein also outlines and discusses a rather comprehensive order of treatment. This includes the removal of local etiologic factors in

7. Fishbein, Jay N.: A Study of Deafness, Eye, Ear, Nose & Throat Monthly **12**:141 (May) 1933.

childhood, such as diseased tonsils and adenoids, and hypertrophied posterior tips of the inferior turbinates, as well as careful examination of Rosenmüller's fossa for probable adhesions. Removal of systemic causes which may originate in the gastro-intestinal, endocrine or nervous system as well as removal of focal infections is recommended. Therapy may include treatment of the systemic diseases, such as hay fever and syphilis, and direct local treatment of the eustachian tube, such as the application of medicaments, politzerization and catheterization, the passing of bougies and applications to the tympanic membrane, such as mechanical massage, the use of vibrators and direct applications to the drum. Electrical adjuvants include diathermy, ultraviolet rays and roentgen rays. Lastly, he recommends lip reading for all patients with an advanced degree of deafness. The family physician should be impressed with the possibilities of the common cold as a factor in the etiology of deafness. Fishbein also mentions the value of routine examinations following the exanthems, since many are complicated by deafness. Repeated examination with an audiometer should be made to follow the course and progress of disease and treatment.

It is doubtful to the reviewer whether some of the therapeutic measures are of much value in most cases of markedly impaired hearing.

Gottlieb [8] reviews the literature and presents theories and proof to substantiate the fact that constitutional deafness does exist. The etiology of this condition is taken up under two heads: the predisposing causes, namely, heredity and environment, and the exciting causes, which are hepatic, pancreatic and endocrine. In every case of nonsuppurative bilateral deafness showing no local pathologic defect, other general objective and subjective abnormalities occur which, when properly correlated, establish their etiology as well as that of the deafness. Gottlieb stated that constitutional deafness is a preventable and curable condition within certain well defined limits. It can be prevented by the carrying out of yearly audiometer tests on all persons above the age of puberty. Any drop below the original level should be immediately investigated by a complete survey, and all abnormalities corrected, if possible. Impaired hearing is curable in the sense that early cases, as a rule, promptly respond to treatment. In cases over five years in duration, complete recovery is not to be expected, although some improvement does occur or the progress is definitely checked.

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8. Gottlieb, M. J.: Constitutional Deafness, Arch. Otolaryng. **16**:814 (Dec.) 1932.

Pohlman [9] demonstrates the importance of separating the real from the unreal phenomena in audition from the standpoint of the diagnosis as well as from the standpoint of proper interpretation of the mechanics of audition. He used a beat frequency oscillator hooked up through an intensity control to an amplifier and loud speaker. This delivers tones of any pitch from the highest to the lowest audible note under definite controlled intensity regardless of pitch. Pohlman first demonstrated that he himself has a perception deafness for high pitched tones. He next checked his experience with that of a conduction-deafened person who showed a raising of the lower tone limit as compared to himself and who heard the higher tones longer than he did. This phenomenon was not real as far as the high tones were concerned because a conduction-deafened person shows an almost uniform loss in the sensitivity for all frequencies with a lowering of the upper tone limit as well as a raising of the lower tone limit. This was not demonstrated because of the perception deafness of the examiner. Furthermore, although the conduction-deafened person is conscious only of the deficiency in hearing low tones, the impairment for the hearing of high tones is nevertheless present. Therefore, one's consciousness of one's disability is not in itself a criterion of the amount of the hearing impairment. When using the spoken voice test, one must consider the interpretative capacity of the patient as well as the intensity and intelligibility of the source. The examiner, to make auditory tests of value, should make the tests on the patient and not on himself. Although most otologists make comparative tests, Pohlman doubts whether one in five really knows how well he himself hears.

Another point to remember is to test only the hearing of the patient and not something else. For speech tests one must eliminate the factor of sight with the possibility of lip reading, and for the fork and other tests the factor of substituting tactile for auditory impressions. The tests should be made on the patient and not on the room; that is, the same room and surroundings should be used when checking progress or verifying the results of previous tests. The test further should not be on the source of sound but on the patient. Changes in the position of the fork or the head of the patient may vary the results. This emphasizes the importance of the standardization of forks and the position of forks urged and preached by one of us (R. S.).

Pohlman also points out that paracusis willisiana is not a true phenomenon, as was shown by von Troeltsche eighty years ago when he found that persons with paracusis did not hear his tuning forks better in a moving railroad train. He also mentions that the Rinne

9. Pohlman, A. G.: Unreal Phenomena in Audition and Their Relation to Test Methods, Ann. Otol., Rhin. & Laryng. **42**:352 (June) 1933.

test was not really invented by Rinne, because it was described three hundred and thirty years ago by the Italian, Caparacci. Pohlman also brings out the fact that a true prolonged bone conduction does occur in the presence of a functional sound conduction system when the external canal is completely or partially occluded or when the drum membrane is loaded. This occurs by true craniotympanic conduction as opposed to the direct cranial conduction found in cases of stapedial fixation. Pohlman demonstrated the relation between air and bone conduction as opposed to the direct cranial conduction found in cases of stapedial fixation. He also demonstrated the relation between air and bone conduction by using the test described by Caparacci in 1603. The latter found he could differentiate conduction deafness from perception deafness by a simple test. The conduction-deafened patient could hear the music of his zither when a rod held between his teeth was in contact with a sounding board, whereas the perception-deafened person could not. Pohlman demonstrated this principle, using an acoustic fan. Persons with a positive reaction to the Rinne test react negatively to the fan test, but persons with a typical negative reaction to the Rinne test react markedly to the fan test. If the patient reports the speech more intelligible to him, then the perception apparatus as far as speech range is concerned is relatively normal. If he reports only an increased loudness with intelligibility the same, then his perception apparatus is also disabled. If he reports no marked improvement, then the sensitivity of the entire perception apparatus is lowered. This test will operate only when bilateral deafness is present, and it is of great importance.

For practical purposes, this test made with a single tuning fork of a frequency between 256 and 512 together with the use of an acoustic fan can give the otologist all the information he needs to make a diagnosis of conduction, perception and mixed deafness in practically all his cases.

Pohlman is an indefatigable worker, investigating constantly not only the anatomy of the mechanism and the physiology of audition, but also the history of medicine with reference to the ear.

Muller [10] introduces a new instrument called the iatrophone for the examination, diagnosis and treatment of chronic deafness. It is an electric interference tone-generator equipped with a quartz crystal. The latter makes it quite constant in frequency to a degree of 1 part in a thousand, and this constant frequency is the pure sinusoidal form of vibration. It is a small portable unit weighing about 16 pounds (7.3 Kg.) and adaptable to voltages of from 100 to 120 and of from 200

10. Muller, Isidor: Demonstration of a New Apparatus for Examination, Diagnosis and Therapeutics of Chronic Deafness, J. Laryng. & Otol. **48**:559 (Aug.) 1933.

to 240 on an alternating current of from 40 to 60 cycles. The price is reasonable. The advantage of this machine is that by means of a special loud speaker connected with the patient's ear, simple tones as well as complicated noises, such as speech and music, can be transmitted.

The hearing is first tested by whispering or, in a very deaf person, by loud speaking. This is followed by the tuning fork tests such as the Weber, Rinne, Schwabach and Gellé tests. The air conduction is then tested with tuning forks of different pitches. Muller uses electromagnetic tuning forks and for several years employed these with an apparatus by which "tone frequent currents" are used. Tone frequent currents are frequencies of electric oscillation. With this every audible tone and tones beyond the range of hearing can be produced almost free from overtones. Furthermore, a continuous tone succession can be generated by electro-acoustical vibration. This apparatus has a range of from 0 to far above the limit of hearing, namely, 20,000 and more vibrations, and these may be used for testing of hearing. Combined bone and air conduction as well as air or bone conduction alone can be determined by bringing the end of the device either quite close to, or inside, the meatus, or by applying it to the mastoid process. The height of the tone as well as its intensity may be varied as required in a more rational way than with tuning forks.

The perception faculty of the auditory nerve can be tested in every way by measuring the length of time in which the central perception of sound is appreciated. Information can also be obtained as to the process of perception, especially in regard to central hearing, namely, the cerebral perception of the auditory phenomena. This is accomplished by testing not only with simple tones but with complicated tones and noises, such as speech and music.

Therapy can be carried out by using the tone frequent currents as simple stimulators without sound. In this way one can excite the auditory nerve without tiring the brain with tones. By varying the intensity of each separate frequency over a wide range of values, one may discover isles of hearing and the gaps between by noting the responses of the subject to the several frequencies and intensities. A method of education is carried out by covering the gaps and widening the corresponding range of hearing by continued and suitable application of adequate frequencies. This kind of tone production is exceedingly adaptable for training the auditory nerve.

Lewis [11] reports several cases of bilateral progressive deafness with tinnitus associated with no known infections or inflammatory or toxic cause, progressing for fifteen to twenty years despite treatments, which

11. Lewis, E. R.: Improved Hearing in Fixation Deafness: Case Reports; Comments, Ann. Otol., Rhin. & Laryng. **42**:135 (March) 1933.

showed striking improvement following the use of systemic measures. The therapeutic measures were essentially a diet high in fluid, protein and fat and low in carbohydrates with alternating periods of chloridizations and iodization, supplemented occasionally with the administration of pituitary from the anterior lobe and thyroid. The object of the therapy was an attempt to produce and maintain chemical alterations in body tissues as a means to effect physical changes in the tympanic structure when the mechanical operations had been interfered with.

The cases reported suggested to Lewis certain clinical and biologic considerations such as the following:

(1) The deafness followed tissue changes which altered the size, shape or texture of bony or ligamentous structures near the oval window;

(2) Clinical evidence suggests gradual increase in degree of these tissue changes;

(3) Bilateral incidence of this type of deafness suggests predisposition to local cellular or tissue changes;

(4) Bilateral incidence of this deafness points to systemic factors whose operation activates the process; some of these systemic factors are commonly associated with puberty and pregnancy;

(5) That attempts to alter the constitution of body tissues have been followed by change in long-standing ear conditions, is shown by the onset of the vertigo, nausea and vomiting, by cessation of tinnitus and by increased hearing acuity; coincident general tissue changes were seen in alterations of body weights, tissue and circulatory tensions, metabolic rates and blood pictures.

Lewis presents a few of the biologic changes and mechanisms which have some bearing on this study. He has done considerable work along these lines for a number of years, but it will require much endeavor on the part of many investigators before one can definitely evaluate the systemic treatment of impairment of hearing.

Smith[12] reviews the literature regarding the effect of noise in industry on the hearing and on general health, and gives the opinions of most of the writers on this subject. He mentions the works of Spooner, Blake, Gottstein, Kayser, Peyser and several others. Peyser stated that continual but moderate noise, if transmitted exclusively by air, has little or no effect on the organ of hearing, but short intense and shrill sound, especially when repeated, injures it in a temporary or permanent way. But simultaneous conduction of noise by air and by the bony skeleton, and above all by the vibration of the ground, finally affects hearing, and seriously so. Cooseman stated that in order for a noisy trade to be harmful, the noise must be intermittent and of a relatively high tone with the worker predisposed to diseases of the ear by the presence of lesions in the nose or pharynx. The report of a

12. Smith, H. F.: Noise in Industry: Its Effect on the Hearing and on General Health, Ann. Otol., Rhin. & Laryng. **41**:1108 (Dec.) 1932.

special committee, the Deutsche Gesellschaft für Gewerbehygiene, for combating impairment of hearing due to industrial noise, brought out several interesting points. Noise can impair hearing by its intensity as well as by its duration. A single strong sound is especially injurious if it occurs suddenly and unexpectedly. Noise injures the auditory nerve endings in the cochlea, which are the most sensitive and vital parts of the organ of hearing. Not every human ear is injured by loud industrial noises. Susceptibility depends on the power of resistance and the state of health of the organ of hearing. Impairment of hearing caused by continuous, moderate, occupational noise comes on insidiously and may not be noticed early. The impairment of hearing is first observed for high notes, and later on as the process extends, the lower tones are affected. To protect the ears, compact plugs of gauze saturated with petroleum or wax should be used. If the floor vibrates, straw, cork or rubber soles may be helpful. People with a definite history of impairment of hearing should consult an otologist before entering employment in noisy industries. A worker who finds that his occupation impairs his hearing should seek different work. Early treatment for impairment of hearing is advocated. Prophylactic measures such as contrivances to suppress noise, as well as relaxation of the worker in quiet surroundings when away from work, are advised.

Hart [13] reports a case of diabetes with a bilateral involvement of the auditory nerve in which the diagnosis was neuritis of the eighth nerve. Diabetes may manifest itself in the ear either as a degeneration of the eighth nerve or as a true neuritis of this nerve. The only means for differentiating these two is by means of the galvanic current test. When the galvanic reaction is absent or nearly absent, a true neuritis of the eighth nerve is probably present. These were the findings in this case. After two months of proper medical care for the diabetes, a significant improvement in the hearing was found. Four abscessed teeth were also removed two weeks prior to the last examination, suggesting the possibility of a focal toxemia. However, since the improvement came on so rapidly and the neuritis as found by the galvanic current was so definite, a focal toxemia did not appear to be the etiologic factor in this case.

McKelvie [14] investigated the hearing in weavers, testing for both the normal speaking and the whispered voice. Of 1,011 persons examined, 24.3 per cent had some form of deafness, 6.7 per cent suffering from impairment of the nerve and 17.6 per cent from some other forms of deafness. The percentage of impairment increased for each decennial

13. Hart, V. K.: Ear Manifestations in Diabetes, Ann. Otol., Rhin. & Laryng. **41**: 1142 (Dec.) 1932.

14. McKelvie, W. B.: Weaver's Deafness, J. Laryng. & Otol. **48**:607 (Sept.) 1933.

period of employment. No case of nervous deafness was found in persons employed less than ten years, but the increase in cases began after twenty years of employment. These results led McKelvie to believe that impairment of hearing in weavers is due to other causes than the usual one of infection via the middle ear. Acute and chronic suppurative otitis media was no more common in weavers than among the general population. The likely causative factors may be the damp, hot and dusty atmosphere of the mills, associated with the noises heard in the weaving shed. The use of a noiseless loom recently perfected would seem to be the best method of prevention of weaver's deafness.

Becker [15] reviews the various bone conduction tests, namely, the Weber, Schwabach, Rinne, Gellé and Bing. The limitations of the Weber test are as follows: Persons with normal hearing may lateralize the sound, probably owing to differences in the thickness of the bone or to other anatomic or pathologic variations in the two sides of the skull. Persons with slight unilateral diseases of the tympanum may be unable to localize the sound. In lesions of a unilateral mixed type of deafness the results may be indecisive, depending on the extent of involvement of each lesion. Some persons with either bilateral acute or chronic diseases of the middle ear or bilateral obstruction of the external ears will lateralize the sound to the better ear, or equally well to both ears. Tuning forks of a higher pitch than 256 double vibrations may give a result opposite to that obtained with the lower pitched forks. In unilateral impairment of hearing, the localization in the normal ear is, per se—without taking into the consideration the history, symptomatology and other corroborative tests—of no positive value. This may also be true in cases of tympanic or labyrinthine diseases.

The limitations of the Schwabach test, according to Becker, are as follows: In unilateral perceptive diseases or bilateral unequal involvement the test is unreliable, as bone conduction may be borrowed from the normal or better ear. The difference in the time that the patient and the examiner hear a tone when a vibrating fork is applied to their respective mastoids may be due to anatomic or pathologic differences in the structure or composition of the cranial bones of the two skulls. The time consumed in transferring the tuning fork from one mastoid to the other weakens or damps the vibrations of the fork. When low pitched forks are used for this test, the vibrations may be mistaken by the patient for sound, and so confuse the result of the examination.

The Rinne test also has shortcomings. Persons with moderate tympanic deafness will give a positive reaction to the Rinne test in spite of a prolonged bone conduction. In old persons with shortened

15. Becker, B. M.: A New Differential Test for Deafness, Eye, Ear, Nose & Throat Monthly **12**:244 (July) 1933.

bone conduction as well as with chronic diseases of the middle ear associated with labyrinthine involvement, the results are indefinite. In exceptional cases the Rinne test may be positive in advanced tympanic diseases as well as negative in marked labyrinthine involvement. The Rinne test with a high pitched fork may be indecisive owing to the hearing of the vibrations by air conduction when the fork is held over the bone.

The Gellé test has its limitations. The compression or rarefaction of the air in the external canal will in itself cause diminished hearing. The natural decline in the intensity of the vibrations of the fork during the examination tends to interfere with the results of the test. The difference in hearing before and after compressions may not be sufficiently pronounced for the patient to distinguish. The test itself has a limited purpose, namely, to determine the stapedial fixity.

Finally, the shortcomings of the Bing test are given. In moderate involvement of the middle ear the vibration of the fork may be heard after the finger is removed from the canal of the ear. In advanced involvement of either the tympanum or the labyrinth, the sound will not recur after closing of the canal.

Becker admits that some of these objections are theoretical and may be obviated, and that the Rinne, Weber and Schwabach tests are of great value. Although each test by itself has its drawbacks, when taken together with a history and functional testing of air conduction, it is important in otologic practice.

Becker reviews some of the general considerations of hearing by bone conduction. He again repeats his personal views and data on various tests, such as the palm test, which he has previously reported several times, and which we have also reviewed. His new test is based on the principle that maximal bone conduction is produced by crossed vibration plus a closed cavity, such as a closed canal of the ear. The greater the volume of air set in motion, the greater the bone conduction. Thus, when the finger is placed in the canal the volume of air set in motion is less than when the palm of the hand is placed tightly over the auricle, producing a longer bone conduction in the latter. The final principle involved in the test is that when the skull is set into vibration these vibrations can be heard in the outer air. Therefore, if a fork is placed on one mastoid, the examiner can hear the vibrations if he holds his ear close to the opposite mastoid but not in contact with it. The increased osseous conduction found in conduction deafness and artifical obstructions varies with different conditions. It is greatest in palm obstructions, less in advanced tympanic lesions, still less in finger obstructions and least in mild cases of conduction deafness.

The test is performed by applying the vibrating tuning fork on the mastoid opposite the ear to be tested. The examiner places his ear

in close contact to the one of the patient being tested. Thus, if the right ear of the patient is tested, the fork is placed over the left mastoid and the right ear of the examiner is applied in close contact with the patient's right ear so as to close the canals of the ear and transform them into a closed cavity. The ear to be tested is thoroughly cleansed first, cerumen, pus or any foreign body which might be present being removed. The interpretation of the test is as follows: In cases of normal hearing, both the patient and the examiner will hear the sound the same length of time. If the patient has perception deafness and the examiner normal hearing, the examiner hears the sound longer, and the severity of the deafness is determined by the length of time the examiner hears the sound longer than the patient. In unilateral or bilateral conduction deafness of the patient, both the examiner and the patient will hear the sound the same length of time, since maximum bone conduction is being obtained owing to crossed vibration and a closed cavity produced by the two ears in contact with each other. As this maximal bone conduction under such circumstances is greater than that obtained in conduction deafness, both the patient and the examiner hear the sound for the same length of time.

The test overcomes the objections mentioned under the other bone conduction tests. It can be performed with all forks capable of transmitting sound to the skull, 128 double vibrations being the best. The sound under all conditions except in advanced perception deafness will always be heard better in the obstructed ear.

Anatomic or pathologic inequalities of the two skulls play no part. No time is lost in transferring the fork, nor is it unnecessarily damped, as the fork is held constantly in one place. Finally, the sound in the obstructed ear is very clear and cannot be confused with mere vibrations.

Certain factors must be remembered in performing the test. The ear of the patient must be thoroughly cleaned. The examiner should not have an impairment of perception. Allowance must be made for the patient's either overstating or understating the hearing time, or for his mistaking mere vibrations for true sound.

The test resembles the Weber test in that it utilizes the principle of lateralization, but the localization is in an obstructed instead of deafened ear. The test resembles the Bing test in that it makes use of an artifical obstruction to make a differential diagnosis. However, it bears the greatest resemblance to the Schwabach test, as it brings out the fact that in perceptive deafness the bone conduction is decreased on the side of the disease, and it compares the bone conduction of the patient with that of the examiner. In addition, it eliminates the defects of the Schwabach test, namely, the inability to rule out hearing of the sound by the normal or better ear while the other ear is being tested, inequalities of both sides of the skull and damping of the fork.

The principle of diagonal resonance or crossed perception was employed and described by Schoen and Goldberger in 1925, and reviewed by one of us (R. S.).[16]

Becker[17] presents a modification of the Bing test. This test is performed by placing a strongly vibrating fork on the mastoid of the ear to be tested or on the opposite ear. When the sound is no longer heard, either the patient or the examiner places the palm of the hand against the auricle of the deafened ear and the vibrating fork is placed on the opposite mastoid. If the sound is heard again, one can rule out conduction deafness, otosclerosis or early or slight involvement of the auditory nerve. If the sound is not heard again, one may conclude that there is marked involvement of the perception apparatus or a mixed deafness in which the portion of the nerve is so predominating that it overcomes the prolonged bone conduction of the accompanying lesion of conduction deafness.

Bing's test consists in placing a vibrating tuning fork on the skull, preferably on the mastoid of the ear to be tested, and inserting a finger into the canal of the ear to be tested when the sound is no longer heard. If the sound again becomes perceptible, the lesion is one of perception deafness, and if the sound is not heard again, the lesion is one of conduction deafness.

The objection to the Bing test is that in advanced tympanic and labyrinthine diseases the sound will not return, but in early cases it may be heard again.

The palm held over the affected ear with the fork on the opposite mastoid produces an optimum bone conduction. This procedure is true because it conforms with certain propositions and proofs regarding bone conduction in general presented by Becker in an earlier paper.[18] The factors in this test are: (1) a closed cavity, (2) increased size of the cavity by the addition of the auricular spaces to that of the auditory canal and (3) crossed vibration. On comparing the prolonged bone conduction produced by tympanic disease and by finger and palm obturation, it is observed that that produced by the palm is greatest both in duration and in intensity when the method described is used. This fact can readily be substantiated by placing the fork on various parts of the skull and using first the finger and then the palm.

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16. Sonnenschein, Robert: Functional Examination of Hearing: Review of the Literature from November, 1924, to November, 1925, Arch. Otolaryng. **3**:71 (Jan.) 1926.

17. Becker, B. M.: The Palm and Fork Test in the Differential Diagnosis Between Conductive and Perceptive Deafness: A Reversal of Bing's Finger and Fork Test, Laryngoscope **43**:456 (June) 1933.

18. Becker, B. M.: Hearing by Bone Conduction, Arch. Otolaryng. **16**:364 (Sept.) 1932.

Becker emphasized certain facts and deductions on which the palm-fork tests are based. The prolonged bone conduction with the palm applied to one ear and the fork placed on the opposite mastoid is greater than that found in any stage of conduction deafness. In otosclerosis without involvement of the intralabyrinthine structures, similar results are obtained. Therefore, when this test is applied and no increase in hearing occurs, one may conclude that the ear is not affected by conduction deafness or by otosclerosis.

The examiner may also make objective comparisons by placing his ear in intimate contact with the patient's temporal bone on the same side as the occluded ear. He not only will hear the sound but will also hear it for about the same length of time as the patient. If the latter hears the sound slightly longer than the examiner, it may be due to natural differences in hearing. When the examiner hears it considerably longer, then not only is the diagnosis of the palm-fork test confirmed, but it is also definite that the patient has an advanced lesion of the nerve. This test is reported not to replace other well established methods of diagnosis, but rather to corroborate them.

The report of the Committee for the Consideration of Hearing Tests,[19] appointed by the Section of Otology of the Royal Society of Medicine in England, is very complete and comprehensive. The subject is taken up in three parts. The first part deals with the physical properties of tuning forks, the influence of auditory fatigue and environment, the description of the various qualitative tests with their modifications, clinical significance and application and the tests for hearing for pitch higher than that emitted by tuning forks. The second part deals with a further investigation of the effect of noise in testing, and contains a report on quantitative tests. Part three deals with audiography.

The specifications submitted for tuning forks included the following requirements: A cross on the fork should mark the place where it is to be struck; overtones should be inaudible within ten seconds after the fork is struck; the pitch audible through air and bone should be the same; the rate of decay (decrement) should be neither too quick for recognition of the end-point, nor so slow as to produce auditory fatigue; after a fork ceases to be heard through bone conduction by the normal open ear in a silent room, it should still be heard for six half intensity periods (a half intensity period is approximately equal to 3 decibels) when held at a distance of a quarter of an inch from the orifice of the ear. A complete description of the tuning forks and recommendations for their proper use are given. A pitch of 128 or 256 double vibrations causes the least fatigue, and notes of higher and

19. Report of Committee for the Consideration of Hearing Tests, J. Laryng. & Otol. **48**:22 (Jan.) 1933.

lower pitch produce more fatigue as they rise or fall. Sounds of high pitch are reflected considerably, especially when the tests are made in a room with hard smooth walls such as those covered with paint, etc. The reflection of sound may be so great as to be heard with the opposite ear, though it is plugged with the finger. Tones below 256 double vibrations are reflected with slight intensity. To avoid errors due to interference which occurs when positive and negative waves neutralize each other, testing should not be carried out too close to a reflecting surface. The rate of decay is minimal if the fork is held lightly with the suspended prongs downward.

The two instruments most commonly used for testing the high tones are the Galton-Edelmann whistle and the monochord. The whistle has disadvantages in that tones may be altered by differences of pressure in the current of air, it is difficult to distinguish between a blow and a squeak and one is unable to estimate bone conduction thereby. The monochord has the advantage that it can be used for vibrations up to about 25,000 per second and also for testing bone conduction. The disadvantage of the monochord is the difficulty in estimating the point where the rub becomes a squeak. The highest notes produced by the monochord are heard better through bone conduction than through air conduction. To eliminate the opposite ear when testing for high tones, the method of allowing a current of water to flow onto a capsule connected by a tube to the ear is preferable to digital pressure and the Bárány noise machine.

A brief but complete description of the classic qualitative tests and their clinical significance is given. These include the Weber, Rinne, Schwabach, Gellé and Bing tests, absolute bone conduction and the Escat test. The committee recommends that these tests form the basis of all methods of testing. None of these tests is complete in itself, as it expresses only the clinical state of the organ of hearing in relative terms. The value of the tests depends to a considerable extent on the experience of the observer and the intelligence and help of the patient. The committee does not recommend the use of the silent room for ordinary tests of hearing; a reasonably quiet room is satisfactory.

The report of quantitative tests includes the tests by speech, both for voice and for whisper. To standardize the voice, otologists should train themselves so their spoken words may always be heard at approximately the same distance, 20 feet, which is adopted as the normal distance by the committee. Hearing should first be tested with both ears open, and then with each ear separately. Isolated words and numbers are recommended at a normal rate of speed, namely, three syllables per second. The tests should be carried out under the same conditions of environment. The committee does not attach great importance to the use of the whisper in testing, as the ability to hear the whisper is

no measure of the ability to hear conversation, since the pitch of the whisper lies between 512 and 6,000 double vibrations and this falls outside the ordinary range of conversation. The ordinary range of the conversational voice lies between 128 and 512 double vibrations for males and between 128 and 1,024 for females. The S sound, however, lies between 4,000 and 8,000 double vibrations per second. The committee does not recommend the use of mechanical speech as produced by the gramophone, because of the difficulty in establishing a standard degree of sound and of eliminating the effects of adventitious noise. Added to this is the fact that conduction by air, bone and cartilage is brought into play when telephone receivers are used.

The three usual methods of performing quantitative tests are given. These should aim at measuring any increase in intensity required to make the patient hear. The committee recommends a standard method for recording the results of tests, by means of the half intensity unit. Forks are being manufactured with known half intensity periods, and when used in the same environment the results are strictly comparable no matter what fork is used. This method is applicable to both bone and air conduction, the results being expressed as the variation from the normal by so many half intensity units.

In discussing the audiometer, the committee mentions two advantages, namely, partial exclusion of extraneous noise and the fact that the source of the sound is at a constant distance from the zero point of hearing. The disadvantages are entirely from the standpoint of the telephone receivers. These, the committee claims, convert the meatus into a resonator having both ends closed instead of one. Second, different degrees of pressure are exerted on the cartilage, displacing the tragus and preventing the full sound from reaching the meatus. Third, insufficient pressure permits leakage of sound, and last, in all cases, cartilage conduction will cause error in the results of testing. The use of the loud speaker eliminates these disadvantages when used in a silent room. When it is used at a considerable distance from the ear, the production of standing waves is apt to interfere seriously with the results. The audiometer at present does not permit the satisfactory testing of bone conduction, as it only records the sum of the hearing by conduction and by perception. The present audiometers as oscillometers are scientifically accurate. However, they fail in their application to clinical otology in that their results differ from those obtained with tuning forks. "The results of testing by tuning forks have been for many years and still are the standard in otology, and until audiometers give results directly comparable with them they cannot be accepted as a standard means of testing. New methods must be evolved from existing knowledge and the committee considers that

future methods of testing must be based on the old established tests by tuning forks."

The committee recommends a graphic method for expressing the quantitative tests which it calls audiography. This presents graphically the results at a glance in percentage of units of loss of normal hearing. Improvement or deterioration of hearing is easily appreciated on retesting. The results will be the same for any given patient provided that the forks used are calibrated. The time required to make the graph is no longer than that taken to record the ordinary qualitative tests. The graph consists of vertical lines representing the pitch of the tuning forks used and horizontal lines representing the half intensity units, numbered from above downward. The upper tone limit is marked on the base line of the chart. To determine any loss of hearing in half intensity units, the following procedure is carried out: For air conduction, forks of 64, 256, 512 and 2,048 double vibrations are recommended. The fork is struck and held at the patient's ear. When he no longer hears the sound, the fork is transferred to the observer's ear; the number of seconds the sound is heard longer by the observer is divided by the half intensity period for air conduction, giving the deafness of the patient for air-borne sound in half intensity units. For bone conduction forks with 256, 512 and 1,024 double vibrations are recommended. The same procedure applies, except that the fork is applied to the mastoid process of the patient and then that of the examiner. The results are placed on the graph, and the half intensity units can readily be expressed as percentage loss of hearing.

The committee recommended the following scheme as a guide to testing deafness: The ability of the patient to hear conversation, first with both ears open and then with each ear separately, is tested. The distance at which the voice is heard and that at which words are understood indicate the loss of compensation. Next, the upper and lower tone limits, the Weber lateralizing test, the Rinne test and air conduction and bone conduction, both in half intensity units, are determined.

Watkyn-Thomas [20] presents his views on the estimation of the high tone limit. The high tone limit has been determined by different methods, such as the use of sirens, whistles and the monochord, with widely different results. It is now generally believed to be somewhere between 20,000 and 30,000 double vibrations per second. The following results show the importance of the high tone limit: With an intact or nearly intact upper tone limit there is generally good hearing for speech, though the lower limit may be affected. Furthermore, the degree of deafness is disproportionately greater when the upper limit

20. In Report of the Committee for the Consideration of Hearing Tests, J. Laryng. & Otolg. **48**:77 (Feb.) 1933.

is affected than with an equal loss at the lower end of the scale. A severe loss of low tones up to *c* 1 with a material loss of high tones can quickly produce complete deafness for the spoken voice.

The tuning forks are used for testing up to 4,096 double vibrations, and the whistle or monochord above that. The disadvantage of all whistles is that bone conduction cannot be tested with them. In addition, the intensity is varied by the force of the air current, and it is difficult to insure a constant force of blast. This applies more to the old fashioned Galton whistle than to the modifications, as these can be attached to the electric pump. The advantages are the portability and the fact that patients find it easier to distinguish between the blow and the squeak. The discussion of the monochord is essentially the same as reported by the committee in part I.

A. Lowndes Yates [20] writes the memorandum on auditory fatigue. "Aural fatigue is a condition in which the effect of a prolonged excitation of the ear by sound becomes more and more feeble although the sound remains constant in intensity and pitch." Fatigue due to sounds which are not present in ordinary life, or to the sounds produced by the tuning fork employed in testing in the normal ear, rarely lasts more than five seconds after such sounds cease. The recovery from fatigue is progressive and is rapid at first.

Auditory fatigue may enter the interpretation of hearing tests. The Weber test may be lateralized to the opposite ear when the other is fatigued, whereas no lateralization would take place if that condition were not present. Also, faulty methods of testing may induce fatigue and alter the results obtained. In certain diseases of the ear, such as impairment of the nerve of toxic origin, fatigue may be easily produced, and its rate of disappearance may be very slow. Meyer, Exner and Mach first drew attention to the fact that hearing did not take place until the sound had fallen on the ear for a short period, and that on its cessation the person continued to hear for a brief period. In a normal fatigued ear these periods are brief, but after-hearing tends to be pro-prolonged, at times to such an extent as to make the normal periods of silence between syllables of words indistinct, making it difficult for the person to interpret words. In this way auditory fatigue may interfere with the ordinary tests for conversational voice. Yates also presents a few principles on the effect of adventitious noise on fatigue.

The pitch which causes the least fatigue seems to be 256 or 128 double vibrations, and notes above or below this cause more fatigue progressively as they rise or fall in pitch. The audiometer may give bad results in testing a patient with tinnitus. The higher notes of the audiometer tend to increase this tinnitus. "It would seem that tinnitus can in some cases be engendered by fatiguing the ear to high pitched

notes of considerable intensity, particularly when the patient's upper tone limit is reduced, and in fact there are a few patients who are so sensitive to high pitched notes that their tinnitus may be produced or augmented by testing for long periods with the monochord, particularly when this is heard by bone conduction."

Auditory fatigue consists of two quite definite factors, the fatigue of the nerve endings of the auditory nerve, and the fatigue of its central connections. Hearing depends on the power of sound to stimulate these auditory nerve endings and the power of such a stimulus to cause a spontaneous desire to listen. The cerebral component of fatigue is very important.

Dundas-Grant [20] presents a memorandum on the physics of tuning forks, and Yates discusses the use of tuning forks and audiography. He recommends to the committee that "half intensity periods" be substituted for seconds as the unit of expression of the Rinne test. He also reviews several principles of bone conduction which have been presented in our reviews of previous years. Yates gives a method for measurement of the intensity of the sound emitted by a tuning fork. His discussion on audiography embodies the same principles as those presented in the committee's report but in a more comprehensive form. A study of the report as well as the memoranda given in the appendix of the report is highly recommended for those interested in the present views on the qualitative and quantitative tests of functional hearing.

The use of the "constant" of damping or decrement devised by Harvey Fletcher of the Bell Telephone Laboratories enables one to obtain curves showing the loss of hearing figured in sensation units, similar to those obtained with the Western Electric audiometer. The decibel is already used as a unit, and we fail to see the advantage in using the half intensity period; in fact, having a new unit will perhaps add an element of confusion. Would it not be better to retain a unit such as the decibel which has become quite well established in otologic literature?

We feel that testing with unaccentuated whisper should not be discarded. Conversation is heard further, and in the short rooms available to most otologists in the large cities it is more practical to employ the whisper, using both high and low pitched sounds (in English the numerals have low pitch except six and seven). Then if the whisper is heard only a short distance, e. g., 1 meter or less, conversation may be tested at from 2 to 5 meters. It should be remembered, however, that in using conversation at less than a distance of 1 meter the sound may be heard via the opposite ear.

Hallpike [20] presents a short critical review of the committee's report. He offers an objection to the advocation of the half intensity unit in referring to differences in sound intensity. This simply adds much

unnecessary confusion, since the Bel system has been well established in America and has a fair measure of support in Germany. (The decibel is one tenth of a bel.) The advantages of the half intensity unit are small. It is important that all otologists should understand each other. Hallpike recommends the support of the Bel system. He also presents objections to the hearing test chart which he claims is too complicated. For audiograms to be used seriously, the chart should be a simple one. Hallpike refers also to persons who show but slight impairment with tuning forks but a considerable inability to understand speech, and vice versa. These cases bring up the question as to what the test for deafness is, tuning forks or speech? This point is not cleared up by the committee's report. The statement made by the committee that audiometers have failed in not agreeing with the results obtained with tuning forks is rather astonishing, as it comes from a body which has devoted several years to exposing the fallacies of tuning fork tests. The committee would be more correct in stating that it is largely due to experience gained with audiometers that the faults of tuning fork tests, as commonly employed, have been made so apparent.

Hallpike suggests the use of a chart which is a modification of that suggested by Wegel. This has two lines, a straight one on top to show the threshold line and a curved one depicting the absolute deafness line (similar to that on our regular audiograms). This shows at a glance the rise above normal of the patient's threshold for the different frequencies, the amount of hearing still available at these frequencies and the loss of hearing in percentage at each frequency. This is the most widely used chart of its kind in existence. Hallpike also shows how one could easily combine the half intensity scale down one side with the usual decibel scale down the other side. Tuning forks then could also be standardized in both the half intensity and decibel units by having both their equivalents in seconds engraved on the fork. In this way every one could use both charts and forks in a manner generally understood.

We are quite in agreement with Hallpike that it would cause confusion in otologic literature to introduce the half intensity unit in view of the fact that the calibration in decibels is already well established.

Wangemann [21] compares the Bezold and Mach theories of bone conduction. Bezold held that bone conduction in all cases, or at least for the lowest tones, was by the ossicular-tympanic route; that is to say, the sound wave was transmitted by bone conduction to the drum

21. Wangemann, Hans: Bone Conduction in Unilateral Deafness, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:603, 1933.

membrane, and then by way of the middle ear and ossicles to the inner ear. On the other hand, according to Mach, the sound waves reach the inner ear directly by way of the bones of the skull to the labyrinth, and after stimulating it, are reflected back to the conduction apparatus. Herzog felt that the mobility of the membranes of both windows, namely, the oval and the round, is the essential fact in labyrinthine stimulation. Only then can there be a flow of endolymph current. This brings to mind the fact that both the Bezold and the Mach theory recognize that there must be some movement in the ossicular chain as part of bone conduction, with this difference: According to Bezold the chain plays a primary or active rôle, but according to Mach, it has only a secondary, passive or retrograde rôle arising in the labyrinth.

The author says that one could visualize the Weber experiment, according to Mach and Herzog, as though there were an obturator in the ear, which interferes with the outflow of sound and also interferes with the drum membrane, ossicular chain and stapedial foot plate. This causes greater pressure of the endolymph current at the round window and, with it, increased intralabyrinth stimulation, resulting in more acute hearing. Wangemann felt that with unilateral deafness it is possible to attack the functioning labyrinth from both sides. Thus, two values would be obtained, one from the deaf and one from the hearing side.

Theoretically, there would be two possibilities: 1. The relation remains about the same, namely, the acuity of hearing following occlusion of the external auditory canal is improved, irrespective of whether one tests the deaf or the hearing side. This result would speak for purely osseous conduction, and would be expected, according to the Mach theory, for then the endolymphatic currents and the interference via the ossicular chain would always be the same. 2. The result is variable so that it could be ascribed only to active participation of the sound-conducting mechanism of the hearing side. Such a result would be in line with the Bezold theory, because here the ossicular chain takes an active part in conduction to the labyrinth during bone conduction.

Wangemann made his tests with the oto-audion in a sound-proof room. The closure of the external auditory canal was accomplished by means of a stopper of some plastic material. He examined four persons carefully with the following findings:

In determining whether unilateral deafness was present, the air and bone conduction of the good ear, as well as the bone conduction of the deaf ear, were tested by the oto-audion. Then the auditory canal of the well ear, or hearing, was closed and the bone conduction curves were again obtained, from both the deafened and the hearing side.

In two of the patients in whom the hearing ear had a normal conduction apparatus, there was a decided improvement in the bone conduction on closure of the external auditory canal. In fact, when tested by way of the mastoid process of the deaf ear it was greater than when tested from the side of the hearing ear. The author thus felt that the tympanic conduction plays an active rôle in the stimulation of the labyrinth by bone conduction. The author found that the prolongation of bone conduction is most definite for the lower tones, reaching upward to about *c* 2 to *c* 3 (1,024 double vibrations).

In cases, however, in which the conduction apparatus of the hearing ear was also impaired, there was only a slight improvement in the bone conduction on closure of the external auditory canal, whether the test was made from the hearing or from the deaf side. This fact shows definitely that the conduction apparatus plays an important part in bringing about activation of the inner ear, at least for the lowest tones. The author, therefore, concludes that in the transmission of sound to the labyrinth by bone conduction, at least for the lower tones, the osseotympanic route plays an important part—an assumption much like that of Bezold. He further feels that Runge's conclusions are justifiable, namely, that the ratio of the craniotympanic (osseotympanic) transmission by bone conduction to craniolabyrinthine (osseous) transmission moves toward the latter the higher the tones are. The author feels that there is a difference between the so-called physiologic and the clinical Weber test; the clinical Weber test is usually employed merely to test the lateralization of sound without plugging the external auditory canal. It seems, therefore, that it is analogous to the Rinne test, and that one should speak of a negative or a positive Weber test.

This is an interesting paper and really confirms in many ways what is already known about the Bing test. In connection with the review of Runge's original paper, the Bing test was discussed by one of us (R. S.) in the January, 1926, number of the ARCHIVES OF OTOLARYNGOLOGY.[16] With the Bing test, in persons with normal hearing or in patients in whom bone conduction has been relatively diminished through an involvement of the perception apparatus, closing the external auditory meatus, whether with water, the finger or any other method, usually increases bone conduction somewhat. If bone conduction is increased previous to the test, there is no further lengthening of it by occlusion of the meatus.

Schmuecker[22] mentions the Stenger test, the Lombard reading test and the funnel and tube test, and grants that they are useful in

22. Schmuecker, Karl: Malingering Tests in Otological Expert Testimony, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:319, 1933.

uncovering simulation of total unilateral deafness. These tests, however, do not give any data regarding the degree of hearing which is present. To uncover malingering of bilateral deafness is difficult and often requires close watching on the part of attendants, neighbors and others in order to catch the simulator off his guard.

Lip reading is often an important factor in uncovering these cases. The person who is hard of hearing or who is actually deaf has usually learned lip reading to a marked degree so that he can understand lip movements, even if no audible sounds are produced.

The simulator of total bilateral deafness, however, has, as a rule, had no experience in lip reading, and, therefore, cannot understand when no audible sounds but merely movements of the lips are made by the person who is testing the malingerer. Furthermore, if a noise apparatus is placed in each ear of the simulator, and he is then allowed to watch the lips of some one who is speaking rather loud, he will be unable to understand, whereas the deaf persons will be able to read the lips quite well.

As has been said, for the detection of simulated total unilateral deafness, the Stenger and other tests are of great value. For the detection of bilateral total deafness, many tricks, so to speak, must be employed in order to catch the simulator off his guard. Schmuecker's suggestions with regard to the lip reading test are very good, but one must remember that many people, even though not very hard of hearing or deaf, do a certain amount of lip reading intuitively and, therefore, all precautions must be taken before a definite conclusion is made.

Schlittler [23] reports a large series of tests for comparative purposes, using *c* 5 (4,096 double vibrations) forks and the *c* 5 sounding rod devised by the late Prof. Karl L. Schaefer and Mr. Wethlo. The tuning forks which he employed vibrated for a period of from seven to eleven seconds, whereas the Schaefer sounding rods do so for forty seconds or more. In fact, the particular sounding rods which were used are supposed to vibrate from thirty-two to thirty-five seconds. All patients were tested at least three times, and the average results were noted. Schlittler found that in ninety-three persons with normal hearing the sounding rod was heard on an average of thirty seconds, and in all these persons the hearing for the unaccentuated whisper was normal. The second group, consisting of seventy persons with moderate and severe forms of hearing impairment of the inner ear, showed in practically all cases that when there was a marked diminution in the hearing of speech, there was also an impairment or decided shortening in hearing the sounding rod. Another group of patients with involvement

23. Schlittler, E.: Experience with the Schaefer Sounding Rods, Ztschr. f. Laryng., Rhin., Otol. **23**:330, 1933.

of the inner ear either alone or in combination with impairment of the inner ear was studied. When there was impairment of pure conduction, the relation between the hearing for speech and that for the *c* 5 sounding rod was not often parallel, but in those persons with relatively poor hearing for the voice, sounding rods were still heard for a long time. In those cases in which there was also involvement of the inner ear, the relationship noted between the disability in hearing speech and the shortening of the time for the sounding rods was again noted. In testing a group of persons with unilateral deafness, it was found (as, of course, could be expected from the fact that high tones are hard to exclude) that merely closing the well ear with moistened cotton does not suffice, and that the use of a noise apparatus in that ear is necessary in order not to hear the *c* 5 sounding rod.

Schlittler's conclusions are that the Schaefer sounding rod, because of its long vibration period, serves very well for obtaining qualitative information concerning the hearing for high tones.

Lisolotte Oberwegner [24] cites a long series of examinations of persons with normal hearing and compares the results obtained with the *c* 5 tuning fork and *c* 5 and *c* 6 sound rods. She calls attention to the fact that it has long been recognized that in impairment of the auditory nerve, besides the reduction of hearing of the upper tone limit, the hearing for all other frequencies is also somewhat diminished. Diminution, of course, is most marked in the higher tones, and diminishes gradually toward the lower tone limit. She says, therefore, that to arrive at upper and lower tone limits, a quantitative determination should be made in order to obtain an accurate measure of the hearing acuity.

Uffenorde (in whose clinic the work for the paper was done) is quoted to the effect that:

> The duration of hearing for the *c* 5 (4,096 double vibrations) pitch compared with other tones shows markedly diminished impairment of the auditory nerve. Often, indeed, the determination of an inner ear impairment can be made only from the diminution of hearing for tuning forks, especially the *c* 5. Let me therefore, from a practical standpoint, assume that the perception apparatus is normal if the duration of hearing for *c* 5 is also normal. Furthermore, when repeated tests with *c* 5 show some improvement, then improvement of the hearing ability occurs.

Oberwegner examined a large series of patients who had been very carefully tested, in order to see whether they had normal hearing. The ear which was not being tested was kept occluded by an assistant's finger covered with a rubber cot, or some other substance was placed

24. Oberwegner, Lisolotte: Comparative Quantitative Hearing Tests on Normal Persons, with c-5 Tuning Fork and c-5 and c-6 Sounding Rods, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:197 (July) 1933.

in the canal, and a shaking motion was produced. This really acted as a noise apparatus, and prevented any hearing by the untested ear. The author bases one of her objections to the use of the sounding rod (the one she employed is not constructed exactly like the one used in America) on the fact that between the handle of the rod and the rod are small leather pads which are likely to move slightly during the vibration of the rod. In the sounding rod perfected by Mr. Eisenhour and the committee of the American Academy of Ophthalmology and Otolaryngology, this difficulty is overcome by having no leather between the metal grip and the sounding rod, but a three point metal contact. In quoting the paper which was published by one of us [25] on the Schaefer sounding rod in 1931, Oberwegner stated that the alloy which was used had a specific gravity of 2.8, thus being one-third lighter than steel. This is perhaps a typographical error, because the specific gravity of the aluminum-magnesium alloy is two-thirds lighter than steel. Oberwegner furthermore states that the transverse tones which are produced by the sounding rod when it is struck last for a few seconds and confuse the person whose hearing for high tones is already greatly impaired. We doubt whether this is a material objection, because these transverse tones last such a short time, whereas the longitudinal vibrations continue for a very long time. The author quotes Prof. Karl L. Schaefer to the effect that he objected to the *c* 5 tuning forks because most forks of that pitch in common use vibrated only from seven to fifteen seconds, and because the very best steel *c* 5 forks did not vibrate more than from twenty-five to thirty seconds. Schaefer believed that the result of the testing of hearing with any instrument is more accurate the longer its period of vibration. He, therefore, felt that the period of vibration of the best *c* 5 forks, namely, from twenty-five to thirty seconds, is insufficient. The author concludes that *c* 5 and *c* 6 forks are quite essential for the proper testing of hearing, and at her suggestion Wethlo perfected a *c* 6 (8,192 double vibrations) sounding rod which vibrates approximately twenty-six and a half seconds. According to Oberwegner, because the sounding rod gives forth an undertone in the first few seconds of vibration, because there are changes in nodal points through the movement of the handle of the *c* 5 sounding rod and because there are considerable differences between the maximal and minimal values in hearing time, the rods show no advantage in practice over the *c* 5 tuning fork.

Truebsbach,[26] also working in the Uffenorde Clinic, published his findings at the same time as Oberwegner. The latter author tested

25. Report of Committee on Tuning Forks and Tests of Hearing, Tr. Am. Acad. Ophth. **36**:415, 1931.

26. Truebsbach, L.: Comparative Quantitative Hearing Tests in Nerve Impairment wth c-5 Fork and c-5 and c-6 Sounding Rods, Arch. f. Ohren-Nasen- u. Kehlkopfh. **135**:208 (July) 1933.

normal persons, but Truebsbach tested those with impairment of the nerve. Truebsbach concludes that in disease of the inner ear, the hearing ability for the *c* 6 sounding rod is quantitatively more diminished than for the *c* 5. He said that the long vibrating *c* 5 fork (about twenty-five seconds) has certain advantages over the *c* 5 sounding rod, because there are no undertones to disturb the hearing; because the quantitative hearing ability for the forks is less than it is for the rods; because there is no disturbance due to the loud tones when the sounding rod is struck, and because there is no possibility of disturbing or changing the nodal points. As to the *c* 5 and *c* 6 sounding rods, the latter is preferable because with it the quantitative hearing ability is reduced more than with the *c* 5. Furthermore, the sound made on striking it is not so loud or disagreeable as with the *c* 5 sounding rod. The author finally concluded that a definite judgment as to whether in otologic practice the *c* 5 tuning fork or the *c* 6 sounding rod is better cannot be rendered at the present time. If the various mechanical and other disadvantages of the sounding rod can be eliminated, then the *c* 6 sounding rod should be chosen; otherwise the long sounding (twenty-five seconds) *c* 5 tuning fork is preferable, for with its findings, a diagnosis of impairment of the auditory nerve can be made.

On a number of occasions one of us (R. S.) has called attention to the work that has been done by the Committee on the Standardization of Tuning Forks and Hearing Tests of the American Academy of Ophthalmology and Otolaryngology. [One of us (R. S.) has been chairman of this committee ever since it was appointed in 1921.] The committee made a special effort to develop the manufacture of tuning forks which would be free from the objections raised against the best steel forks. In the first place, steel, unless properly protected, tends to rust, and the pitch thereby is changed. On the other hand, when forks are nickeled, the nickeling finally tends to peel, and the loose pieces of metal, when the fork is vibrating, give adventitious sounds. In cooperation with Mr. B. E. Eisenhour of Riverbank Laboratory, Geneva, Ill., a series of forks, made of a magnesium-aluminum alloy, was perfected. The first ones, however, although they did not rust, oxidized and changed color. By increasing the amount of aluminum in the alloy, the forks were not only rustproof but tarnishproof, and very light in weight (the alloy had a specific gravity of about 2.8, or was one third as heavy as steel). When Professor Schaefer and Mr. Wethlo in 1930 devised the *c* 5 sounding rod, Mr. Eisenhour was induced to use the same rustproof, tarnishproof magnesium-aluminum alloy for sounding rods as that from which the forks were made. Professor Schaefer's sounding rod vibrated for about forty seconds. The ones that we have vibrate, when properly struck, from seventy-five to ninety seconds. We know the overtones of high pitch forks like the *c* 5 or *c* 6

fork do not act as a disturbing element because they are so high that most persons cannot hear them at all. It is only with the lower pitched forks that overtones are confusing to the patient. It is the longitudinal vibrations of the sounding rod which give one the tone with which one is testing. The transverse vibrations will produce an undertone of short duration, and this cannot be considered a serious objection. The undertone lasts but a few seconds, and is usually damped by the grip which is placed at the exact midpoint of the rod. On the sounding rods used by Oberwegner and Truebsbach there are small leather pads between the metal grip and the sounding rod, and these pads may move during vibration and thus change the nodal point; but with the rod perfected by Mr. Eisenhour, this objection is eliminated because of the three point metal contacts by which the grip is attached to the sounding rod without any leather pad. We have found the Schaefer sounding rods as perfected in this country exceedingly valuable, especially the *c* 5 sounding rod, and we believe it is preferable to the *c* 5 fork. It is extremely difficult to get a *c* 5 (4,096 double vibrations) fork which has a period of vibration of more than from twenty-five to thirty seconds. In fact, most of them have a much shorter period of vibration than this. So far as the *c* (8,192 double vibrations) sounding rods are concerned, the ones previously perfected by Mr. Eisenhour vibrate only fifteen seconds, but the newest ones vibrate on the average from thirty to thirty-five seconds, therefore even longer than those made under the instructions of Oberwegner, namely, twenty-six seconds. We are thoroughly convinced that the sounding rod will prove a most valuable addition to our hearing test instrumentarium, and that it will give eminent satisfaction to the otologist who uses it.

Presenting the end of the rod to the ear, one hears only the sound of the longitudinal tone, but if the rod is held sideways, one hears the undertone. Lightly touching the rod with the finger (for one instant) about one-fourth the distance from either end eliminates transverse tone but does not influence the longitudinal tone appreciably.

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Abstracts from Current Literature

Ear

ASEPTIC OTOGENOUS SEROUS MENINGITIS: CURE. LACROIX, Rev. d'oto-neuro-opht. **10**:742 (Dec.) 1932.

The case reported is that of a young girl in whom, in the course of chronic otitis, meningitis developed. The cerebrospinal fluid contained 150 cells and 70 cg. of albumin. No organisms were found on culture. Immediate radical mastoidectomy with wide exposure of the dura was done, and a large cholesteatoma and a perisinous abscess were revealed. A fixation abscess was produced, and rapid recovery followed. Lacroix emphasizes that the operative results are better when the fluid is clear and the cellular elements are intact, and that prompt and thorough operation is important.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

CERTAIN PARTICULARS OF LABYRINTHINE REACTIONS IN THREE CASES OF TRAUMATIC TEMPORAL LESIONS. G. PORTMANN and P. DELMAS-MARSALET, Rev. d'oto-neuro-opht. **11**:23 (Jan.) 1933.

In an attempt to clear up some points regarding the cerebral terminations of the vestibular pathways, three cases of injury to the temporal region are reported. Many writers attribute anomalies in the vestibular reactions in cases of cerebral tumor to the secondary effects of hypertension. In traumatic lesions such an explanation cannot be valid. In all three cases vestibular disturbances appeared some years after the injury; in one they were accompanied by epileptic attacks and deafness, and in two caloric and rotatory tests gave normal reactions. In one case there was spontaneous deviation in walking with dissociation in the results of the rotatory and caloric tests. In the other two cases, after rotation to the right, deviation in walking was to the left, and after rotation to the left, deviation was to the right, constituting an inverse reaction. The test of provoked deviation in walking was evidently not made in the first case.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

INCOMPLETE FACIAL HEMIATROPHY OF SYMPATHETIC ORIGIN. G. WORMS, Rev. d'oto-neuro-opht. **11**:99 (Feb.) 1933.

Following the healing of an extensive mastoid wound, a young man began to experience paresthesia of the face on the side of the wound, and within three months facial hemiatrophy was noticed. The atrophy was limited to the soft tissues and was accompanied by localized sweating of the forehead, cheek and parotid region, hyperesthesia of the two upper branches of the fifth nerve and increase of the nasofacial reflex. Increase of retinal arterial tension, absence of the oculocardiac reflex, enophthalmos and two bald spots on the temporal region on the same side were observed. Worms believes that these troubles arose from a disturbance of the vegetative system, caused by irritation from the mastoid scar. In support of the assumption that the sympathetic system was involved, another case is reported in which the condition was not due to trauma but followed repeated attacks of sympathalgia. Neither case presented typical trophoneurosis, involving both the soft and the skeletal parts. Cases of facial atrophy of traumatic origin have been reported by Weekers, Leriche and others. In certain cases removal of the superior cervical sympathetic ganglion has been followed by good results.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

MORPHOLOGIC PICTURE OF CRISTA ACUSTICA DURING THE STATE OF STIMULATION. K. WITTMAACK, Arch. f. Ohren- Nasen- u. Kehlkopfh. **135**:41 (May 17) 1933.

Wittmaack says that Huizinga's observation that a typical nystagmus can still be evoked by galvanic stimulation after destruction of the cupula and the epithelial border, which indicates that a galvanic stimulation acts directly on the peripheral terminations of the nerves, is in keeping with his own theory of tonus and is corroborated by some of his experiments. These facts indicate that the shifting of the fluid, which according to his theory takes place in the cupula during the act of stimulation and which causes the drop in pressure necessary to produce the stimulus, also occurs along minute liquor canals, through the epithelial border into the subepithelial tissue. With this in mind he studied the subepithelial tissue region during a state of excitation more closely than he had previously done and under maximum magnification. In the microscopic examination of corresponding regions of two cristae which were in opposite states of stimulation during decapitation of the animal, a marked difference in the condition of the subepithelial tissue was noticed. The current was too weak to permit permanent injury. In the crista compressively stimulated by cathodal excitation there was a much greater accumulation of fluid in the subepithelial tissue, and therefore a much greater distance between the individual cells, than in the crista depressively stimulated by anodal excitation. On the whole the cells of the former appeared a little less voluminous, i. e., more compressed, than those of the depressively stimulated crista. This was in agreement with the condition of the cupular framework itself, the fibers of which appeared much more loosened on the depressively stimulated than on the compressively stimulated side. Analogous though slightly less pronounced differences in the subepithelial tissue are found in caloric and rotatory stimulation. In the crista stimulated with cold water the ascending nerve fibers in the subepithelial tissue appeared wider and paler than those in the crista stimulated with hot water, the first case corresponding to a direct depressive action, and the second case to a direct compressive action, on the nerve endings, as indicated by Huizinga.

EDITOR'S ABSTRACT.

NEURINOMA OF THE ACOUSTIC NERVE. W. HACKEL, Beitr. z. path. Anat. u. z. allg. Path. **88**:60, 1932.

The gross and microscopic structure in twelve cases of neurinoma of the nevus acusticus is described and illustrated by sixteen photographs. Three principal types are distinguished. The first, unripe, variety is composed of large oval nuclei lying in a homogeneous substance. The second variety corresponds to the classic description by Verocay and shows long, rod-shaped nuclei and very fine fibrils. The third type of neurinoma is characterized by small round nuclei and a definite reticular stroma. There may be xanthomatous elements in all three types. The variation in the size and structure of the nuclei observed in these tumors is not an indication of malignancy, but is due to degeneration.

HELLWIG, Wichita, Kan. [ARCH. PATH.]

INJURY TO EARS DUE TO NOISE AND VIBRATION. J. TEMKIN, Monatschr. f. Ohrenh. **67**:257 (March); 450 (April); 527 (May); 705 (June); 823 (July) 1933.

Temkin discusses at length the results of his five year study of occupational aural injuries due to noise and vibration. The investigation included the following occupations: weaving, boilermaking, scythe forging and other metallurgic work, the manufacture of chocolate, the testing of airplane motors and stenography. After discussing the character of the noises and vibrations encountered in each of the occupations and the methods used in analyzing and measuring them, and briefly describing the methods of clinical otologic examination employed, the author gives the results of the investigations. He first takes up the frequency and

degree of deafness found in each of the occupational groups and the clinical character of the aural changes typical for each of these groups. The significance of personal factors, such as age, periods of rest, infectious diseases, constitution and heredity and, particularly, pathologic changes in the sound-conducting apparatus (various forms of otitis), in determining variations of the typical aural changes of each group is stressed. The subject of decrease in auditory acuity during the course of the working day is taken up. The author next considers the rôle of vibration and bone conduction in the production of aural changes, and presents a searching study of the mechanism by which noise and vibration cause aural injury. The changes in the vestibular apparatus are also discussed. Prophylactic measures for the protection of the ear should be along three lines: general hygiene, governmental industrial regulations and technical reconstruction. Along the first line rational and healthy recreation should be cultivated; alcoholism and so on should be combated. Along the second line shortening of the working day to seven hours and of the week to five days, regular monthly leave of absence and rotation of workers between noisy and noiseless occupations within the same industry are suggested. The length of exposure to noise in one day is of utmost importance. Persons with labyrinthine disease, congenital baryecoia or chronic purulent otitis media with signs of labyrinthine injury should not be admitted to noisy occupations. In other forms of chronic otitis media, admission to noisy occupations must depend on the individual functional condition and so on. Persons with purulent otitis should not be admitted to noisy occupations in which there is also vibration, regardless of the functional condition of their ears; not only is their hearing injured, but the suppurative process is exacerbated. Technical improvements should consist in the reduction of resonance in the buildings and substitution of noiseless processes for noisy ones wherever possible. A special platform for the reduction of vibration is described. Elimination of vibration permits the use of sound dampers and antiphons, which are of no use while vibration continues.

EDITOR'S ABSTRACT.

TREATMENT OF CHRONIC OTITIS WITH MEDICATED FOAM-PRODUCING PENCILS. M. SCHARFSTEIN, Monatschr. f. Ohrenh. **67**:978, 1933.

The pencils used by Scharfstein for treatment of the ear, which have been frequently employed in gynecology, are composed of a substance which in contact with fluid (mucus) produces a long-lasting foam. Under the pressure of the carbon dioxide which is formed, the foam spreads through the mucus into all the folds and crevices of the mucosa, thereby bringing the medicament which is contained in the pencil into intimate contact with all portions of the mucosa. The pencils can be obtained with various medicaments: zinc sulphate, salicylic acid, silver protein, silver nitrate and tannic acid. The author breaks the pencil into pieces small enough to be inserted into the middle ear through the perforation of the tympanic membrane and inserts two or three pieces near the antrum or attic following an air douche and drying of the ear. The perforation is closed with cotton. If the production of foam is very great he repeats the process several times at one sitting. Large quantities of mucus are discharged with the foam. After the foam has dried the mucosa is hyperemic for from two to three hours. Six of seven patients with chronic suppurating otitis media were cured or improved by this treatment. In two cases of chronic otitis with cholesteatoma only the nature of the secretion was changed. As water is contraindicated in cases of cholesteatoma, the use of this therapy seems indicated.

EDITOR'S ABSTRACT.

SO-CALLED HYDROPS TYMPANI EX VACUO. G. CLAUS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:162 (June 8) 1933.

On the basis of refractometric investigations, Claus states that fluid in the dependent parts of the tympanic cavity, the upper level of which is visible through

the delicate tympanum as a dark concave line, has a higher albumin content than the blood serum in the majority of cases. In these cases the fluid is undoubtedly an exudate. In the few cases in which the albumin content of the tympanic fluid is less than that of the blood serum, it is nevertheless over 4 per cent, which is considered the boundary line between transudate and exudate, and the fluid must therefore be classed in the latter group. From this point of view it appears that in none of the cases observed was the effusion of fluid the result of a negative pressure, and therefore the condition could not be considered so-called hydrops tympani ex vacuo. This is in agreement with the clinical observation that even in the cases showing a tympanic fluid of low albumin content air could reach the middle ear through the eustachian tube, and therefore hermetic occlusion of the tube—the prerequisite for the development of hydrops tympani ex-vacuo—never occurred.

EDITOR'S ABSTRACT.

ACUTE AND CHRONIC OTITIS MEDIA WITH ENDOCRANIAL COMPLICATIONS AND PREGNANCY. F. WIEGMANN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:115 (May) 1933.

Wiegmann reports two personally observed cases of otogenous endocranial complications in pregnancy. In the first case the patient was in good health until the development of an acute otitis media following tonsillitis in the fourth month of pregnancy. Despite complications consisting of an epidural abscess, a metastatic cerebellar abscess with meningitis and later jaundice and hematuria, the patient's resistance remained strong and drainage of the suppurating foci was followed by rapid and complete recovery. The second case concerns a woman with chronic otitis media, which was exacerbated toward the end of the second and third pregnancies. Endocranial complications consisting of an epidural abscess and a labyrinthine fistula, originating from a putrid cholesteatoma, were found at an operation performed five days after parturition. Recovery was slow; the patient required ambulatory treatment five months after the operation. In neither of these cases was any influence of the otogenous complication on the course of the pregnancy, or any reason for interruption of the latter, observed. The effect of the pregnancy on the otogenous complications in the two cases was radically different. Lundwall found greater resistance to infectious diseases in the healthy pregnant woman, owing to an increased function of the reticulo-endothelial system. Increased mortality is due to a blockade of the reticulo-endothelial system. The first patient was a healthy woman with an excellent reaction capacity, which manifested itself in the rapid recovery after drainage of the disease focus. The second patient was an undernourished woman with a constitutionally inferior capacity for reaction, who was unable to compensate the extra demands made on her toward the end of pregnancy.

EDITOR'S ABSTRACT.

OTITIS MEDIA IN SCARLET FEVER. F. RADE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:126 (May) 1933.

Rade presents a statistical study of the otitis media of scarlet fever from a survey of 2,835 cases of scarlet fever. The incidence of scarlet fever was greatest in March and October, whereas most of the cases of otitis occurred in February, April and June. Years of large epidemics were marked by a high percentage of otitis. The percentage of cases of otitis was parallel not with the mortality but with the morbidity. In 12.64 per cent of all patients with scarlet fever otitis developed; in 31.5 per cent of the cases the otitis was bilateral. Otitis developed in 28.64 per cent of the children between 2 and 3 years of age, but in only 3.12 per cent of the children between the ages of 12 and 14. In most cases the otitis appeared early in the course of the scarlet fever. In 23.6 per cent of the cases it appeared during the first three days, and in 17.2 per cent of the cases during the fourth week as a second manifestation of the disease. Only a small number of

cases of otitis developed between these two periods, and these usually resulted not from the scarlet fever itself but from its complications. Mastoiditis occurred in patients who no longer had any complications except otitis and in whom the otitis had developed at the beginning of the disease. Of the 41 patients who died of scarlet fever, 8 had otitis, but only 1 died from the sequelae of the otitis.

EDITOR'S ABSTRACT.

Pharynx

DIPHTHERIA-LIKE ORGANISMS FROM NASOPHARYNX. M. M. BARRATT, J. Path. & Bact. **36**:369, 1933.

A group of aberrant diphtheria-like organisms have been described which, while resembling Corynebacterium diphtheriae, differ from it in certain cultural characters, most notably in their capacity to liquefy gelatin. The lesions produced in guinea-pigs by intradermal injection of the organisms are more papular and pustular than those following the injection of typical C. diphtheriae. Diphtheria antitoxin does not prevent these cutaneous lesions and even in large doses does not save the life of the animal if more than one or two minimal lethal doses are given subcutaneously, or with some strains even if only one is given. Toxic filtrates produce lesions of the skin which do not resemble those of C. diphtheriae. Diphtheria antitoxin, unless given in larger doses than from 80 to 100 units to one minimum lethal dose of toxin, does not protect the animal from death. The aberrant strains differ further from C. diphtheriae in being pathogenic for rats by intraperitoneal inoculation. When the characters of the aberrant strains differ culturally or biologically from those of C. diphtheriae, they approach, if not completely, those of Corynebacterium pseudotuberculosis-ovis (bacillus of Preisz-Nocard). Although evidence is lacking of the association of the particular strains studied with severe cases of clinical diphtheria, the occurrence in the human nasopharynx of diphtheria-like organisms, the pathogenic effects of which in the guinea-pig are not prevented by diphtheria antitoxin, has been established. Therefore, the possibility that strains of this type play a part in the causation of cases which do not respond favorably to antitoxin cannot be altogether ignored.

AUTHOR'S SUMMARY. [ARCH. PATH.]

HEMATOGENOUS INFECTION OF THE TONSILS. C. KRAUSPE, Virchows Arch. f. path. Anat. **287**:139, 1932.

Previously reported results of experimental work had led the author to conclude that the tonsils may be involved by way of the blood stream and that the resulting involvement is often not to be readily distinguished from that which occurs by way of the exposed surface of the tonsil. The present communication is based on the histologic examination, in serial sections, of the tonsils of eleven children and fourteen adults who died of a variety of acute infectious diseases. The tonsils of twenty-five children whose death was not due to acute infection were also examined, but somewhat less thoroughly. In most of the cases of infection, especially in children, the tonsils revealed inflammatory reactions that the author interprets as the result of hematogenous localization. The earliest stages were noted in and about the arterioles of the follicles and in the subepithelial capillaries. Necrosis and abscesses were frequently observed. The bacteria could usually be demonstrated in the lesions. Streptococci were the most important invading organisms in children. In both children and adults, localization of bacteria in the subepithelial capillaries led to inflammatory foci not to be distinguished from primary crypt infections. Involvement of the tonsils was seen in one case of typhoid and one of bacterial glomerulonephritis.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

Larynx

STROBOSCOPY IN LARYNGOSCOPY. O. HEYMANN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **136**:64 (Aug. 24) 1933.

Heymann says that the question of the real or apparent immobility of a vocal cord in the stroboscopic picture is of no clinical significance, because stroboscopy concerns itself with a comparison of the movement of both vocal cords. Every deviation from the synchronous vibration of the vocal cords is of diagnostic importance. In 6 of 50 patients with normal larynges there was a slight retardation of the left vocal cord. The author performed stroboscopic examinations in 136 cases of functional and organic laryngeal disturbances, including change of voice, phonasthenia, acute, subacute and chronic laryngitis, nodules of the vocal cords, recurrent paralysis, laryngeal tuberculosis and hypopharyngeal carcinoma. In functional vocal disturbances the total positive results do not deviate much from the normal; about a third of the cases remain unexplained. In about one half of the cases of organic laryngeal disease the stroboscopic image supplemented the laryngoscopic observations. In one fifth of the cases examined stroboscopy gave negative results. The pictures showed differences of level in 6 per cent of the cases, apparent immobility of one cord in 10 per cent, unequal vibration in 22 per cent and retardation of the left vocal cord in 22 cases and of the right cord in 5. Especially valuable results were obtained in early or suspected laryngeal tuberculosis. In 7 of 8 cases of manifest laryngeal tuberculosis pronounced differences of vibration (in 3 cases immobility of one vocal cord) were seen stroboscopically. The stroboscopic pictures in some cases reflected the regression or exacerbation of the tuberculous process. This was also observed in chronic laryngitis and in cases of papillomas of the larynx. The author thinks that in a demonstrated case of laryngeal tuberculosis with a negative stroboscopic result, involvement of the vocal cords may be excluded, for the time being. Stroboscopy aids in determining the localization of the process and the time at which the vocal cords become involved. While negative results are not a differential diagnostic proof of a non-tuberculous laryngeal disease, continuous laryngostroboscopic control is valuable for prognosis in cases of suspected laryngeal tuberculosis. The author thinks that stroboscopy should supplement laryngoscopy, especially in organic laryngeal diseases, d spite the many difficulties involved. EDITOR'S ABSTRACT.

PHONATION WITH FALSE VOCAL CORDS, DYSPHONIA SPASTICA AND PHARYNGEAL MUSCLES. A. RÉTHI, Monatschr. f. Ohrenh. **67**:572 (May) 1933.

Réthi presents a detailed discussion of his studies of the nature and cause of so-called false vocal cord voice and dysphonia spastica. The false vocal cord voice is produced by substitution of an increased function of the stylopharyngeal muscle and its accessory, the palatopharyngeal muscle. Spastic dysphonia is a convulsive manifestation of the same mechanism that causes the false vocal cord voice. The cure of spastic dysphonia and of a false vocal cord voice existing in the presence of true vocal cords capable of function is achieved by interrupting the continuity of the laryngeal portion of the stylopharyngeal muscle. A harmless and simple procedure for this operation is described. EDITOR'S ABSTRACT.

SURGICAL TREATMENT OF CONGENITAL LARYNGEAL DIAPHRAGM. G. KRIEGSMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:218 (June 8) 1933.

In a woman, aged 25, who had always had a hoarse voice and who complained of dyspnea, Kriegsmann found a membrane extending from the front of the glottis and obstructing almost two thirds of it. The grayish-white, somewhat translucent membrane did not affect the motility of the vocal cords. Following excision of the membrane by cutting along the free edges of the vocal cords an improvement in breathing was observed, but the voice was completely aphonic and a fibrinous

exudate containing some cellular elements appeared in the glottis which had to be removed by curettage. The exudate continued to reappear despite all efforts to prevent it, and a membrane of almost the same size as the first one developed within twenty-three days. An incision of the new membrane between the vocal cords and coagulation of the surfaces of the wound by surgical diathermy cleared the glottis and relieved the dyspnea, but five weeks later a new membrance, slightly less extensive than the original one, had formed. The voice was still aphonic, and the dyspnea had returned. After a twice repeated sharp incision of the membrane in the median line, the tendency to adhesion and reconstitution of a membrane was somewhat restrained, and the patient was dismissed with fair results. Epithelization of wound surfaces in places not previously covered with epithelium is hard to obtain. Surgical therapy for a congenital laryngeal diaphragm is recommended only if urgent indications exist. If the membrane is thin toward the back, as in this case, a median incision of the thin portion is advised. If the membrane is thick, the larynx should be opened to permit postoperative treatment after division of the membrane and until the final result can be seen.

EDITOR'S ABSTRACT.

Nose

ACUTE ETHMOIDITIS. CHARLES GILMORE KERLEY, Arch. Pediat. **50**:136 (Feb.) 1933.

A case of acute ethmoiditis with exophthalmos is presented, and the necessity of early diagnosis and treatment is stressed.

WADDELL, JR., University, Va. [AM. J. DIS. CHILD.]

DEVELOPMENT, STRUCTURE AND CHANGES IN THE FORM OF THE HUMAN NASAL SEPTUM IN THE FETAL PERIOD. K. HILLENBRAND, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:1 (May 17) 1933.

Hillenbrand investigated the normal development, anatomy and changes in the form of the nasal septum in ninety-three fetuses and newly born infants with the aid of serial sections and reconstruction models. The cartilaginous nasal septum, the vomer and a part of the intermaxillary bone develop from the median frontal process. They are present in the fetus between 2 and 3 months of age. The fetal cartilaginous septum in its lower portion is held in a groove between the lateral lamellae of the vomer. Anteriorly it lies immediately on the intramaxillary bone. The ossification of the septum commences in the eighth fetal month; it proceeds from the sphenoid sinus. The vomer does not receive its base until later in fetal life. In all specimens a bilateral, physiologic accessory ridge of the cartilaginous septum was noted, which is described in detail. Of the ninety-three noses examined, fifty-seven (61.3 per cent) had an asymmetric septum; in twenty-four cases this asymmetry resulted predominantly from changes of the vomer and in the other thirty-three from changes of the cartilaginous septum. The author discusses the pathogenesis of asymmetry of the vomer, deviations of the cartilage, the crest and ridge formation. He discusses the rôle of the physiologic accessory ridges in the development of clinical ridge formation and compares the existing theories for the development of deformities of the nasal septum. From the results of Schwartz's investigation on twins and his own studies the author assumes that most deformities of the septum are determined by the genes.

EDITOR'S ABSTRACT.

VOLUME OF THE MAXILLARY SINUS FROM THE PHYSIOLOGIC AND FROM THE PATHOLOGIC STANDPOINT. J. TREER, Monatschr. f. Ohrenh. **67**:681 (June) 1933.

Treer studied the volume and the rate of drainage of the maxillary sinus by means of injections of iodized poppy seed oil. In persons between the ages of 25 and 60, the volume of the maxillary sinus under normal conditions varies between 5 and

12 cc. Under pathologic conditions the volume of the maxillary sinus in 58 per cent of the patients was only from 0 to 4 cc.; sinuses of this size were not found at all under normal conditions. Sinuses with a volume of from 5 to 7 cc. were found in 20 per cent of the normal subjects and in 27 per cent of the persons with pathologic conditions. Sinuses with a volume of from 8 to 12 cc. were found in 80 per cent of the normal subjects but in only 15 per cent of those with pathologic conditions. This shows that the measurement of the unobstructed volume of the maxillary sinus is a measure of the extent of the pathologic process existing in the sinus and is therefore of practical value. Complete drainage of the injected iodized oil from the normal sinus was effected in one week in 16.1 per cent of the subjects, in two weeks in 61.3 per cent and in several weeks in 22.6 per cent. The drainage of sinuses obstructed by polyps in the middle nasal meatus was effected in the first week in 12 per cent of the subjects, in the second week in 36 per cent and in several weeks in 52 per cent. The author thinks that in view of its poor drainage conditions the maxillary sinus may be considered a physiologic defect; its chronic inflammation may be attributed to the highly deficient drainage.

EDITOR'S ABSTRACT.

COMPARATIVE CLINICAL EXAMINATIONS OF MAXILLARY SINUS. J. TREER, Monatschr. f. Ohrenh. **67**:841 (July) 1933.

Treer compared the following methods of examination of the maxillary sinus: irrigation, roentgenoscopy with and without a contrast medium, determination of the volume and rhinoscopy. He concludes that a negative rhinoscopy is a more reliable finding than any other. Only a positive rhinoscopy requires further examination. The extent of the pathologic process in the sinus can be best determined by measuring the volume (free space) of the sinus. If the volume is from 0 to 4 cc., surgical opening of the cavity is always indicated; if it is from 8 to 12 cc., further interventions without special indications are superfluous. If the volume varies between 5 and 7 cc., then the positive or negative rhinoscopic results must determine conduct. Exceptionally, measurement of the volume of the maxillary sinus is indicated in a case with negative rhinoscopy; in that case, if the volume approaches the lower normal limits, the cause for this must be investigated.

EDITOR'S ABSTRACT.

OSTEOMA OF THE FRONTAL SINUS. E. KNAPP, Monatschr. f. Ohrenh. **67**:903 (Aug.) 1933.

Knapp reports a case of osteoma of the frontal sinus in a girl, aged 15. The clinical symptoms were a visible flat protuberance above the root of the nose and adherent to the bone, a small palpable protuberance on the inner canthus and next to it on the orbital margin a small pulsating tumor. The tumor of the frontal sinus was diagnosed roentgenologically. At operation it was seen that the osteoma had broken through the anterior wall of the frontal sinus. The pulsating tumor was a mucocele. The osteoma had also perforated the central and inferior parts of the posterior wall of the sinus and had become adherent to the dura. In an attempt to isolate the tumor at this point, the dura was ruptured. The operation was terminated without removal of the tumor. Meningitis did not result.

EDITOR'S ABSTRACT.

RHINOGENOUS RETROBULBAR NEURITIS. T. VON BAJKAY, Monatschr. f. Ohrenh. **67**:907 (Aug.) 1933.

Von Bajkay states that disease of the posterior ethmoid cells and the sphenoid cells is the most frequent cause of rhinogenous retrobulbar neuritis. The disease process may spread from the sinus into the orbit through the osseous wall either by necrosis of the osseous wall or directly through the medullary spaces of the intact bone, or it may spread via the blood or lymph channels. In rare instances,

the inflammation of the ethmoid sinus may give rise to contralateral neuritis. Such a case is cited by the author. Retrobulbar neuritis occurs suddenly, and the visual disturbance assumes a severe form from the start. The first symptom is enlargement of the blind spot; later peripheral restriction of the visual field, central scotoma, amblyopia and amaurosis may occur, depending on the degree of pathologic change in the optic nerve. In cases in which the diseased sinus contains no secretion, diaphonoscopy and roentgen examination of the sinus are required for diagnosis. If there are no macroscopic changes in the sinus mucosa it should be examined microscopically, as microscopic changes may spread to the bone and the adjacent regions. If rhinologic examination gives negative results and the patient is seen soon after the appearance of the visual disturbance, diaphoretic treatment associated with inhalation is usually successful. Cocainization of the nasal mucosa improves the visual capacity by decreasing the edema of the optic canal and improving the circulation. The sinuses are opened if the rhinologic examination gives positive results and even when it does not if an ophthalmologic indication exists and other causes can be ruled out. Among forty cases of retrobulbar neuritis in which the ethmoid and sphenoid sinuses were opened, rhinologic examination gave positive results in twenty-two instances and negative results in eighteen. In both of these groups operation produced a complete cure in those patients who entered the clinic from three to four days after the onset of the visual disturbances, but in those who entered between the eighth and the fourteenth day after the onset of symptoms only a partial restoration of vision could be obtained. In eight cases in which from three to four weeks had passed since the onset of the symptoms no improvement was obtained.

EDITOR'S ABSTRACT.

BARRIER FUNCTION OF THE NOSE. W. WOJATSCHEK, W. UNDRITZ and K. DRENNOWA, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:191 (June 8) 1933.

Wojatschek and his associates studied the barrier function of the nasal conchae in human beings and dogs by injecting various substances subcutaneously, intravenously and into the conchae and comparing the results. Epinephrine, sodium iodide, sodium bromide and dextrose solutions were used, and, in animal experiments, also some colloidal dye solutions and some suspensions of india ink and soot. The barrier of the nasal concha is composed of a labyrinth of blood vessels, a strongly developed network of lymph vessels and the reticulo-endothelial elements of the nasal concha itself. The barrier constituted by the lymph system of the nasal conchae must be considered, not apart from, but in connection with, the regional lymph channels and lymph nodes. The substances introduced into the nasal conchae first come in contact with the reticulo-endothelial elements of the conchae. Then a portion of the injected substance, after passing the vascular endothelium barrier, penetrates directly into the blood stream; the other portion penetrates the lymph channels of the concha and from there reaches the lymph nodes, where it passes through the filter of the reticulo-endothelial elements of the nodes. In man with his extensively developed cavernous tissue, the hematogenous route is the main route by which fluids are conducted away from the conchae; therefore injections into the conchae resemble intravenous injections. In the dog, on the other hand, the main mass of fluid enters the lymph channels, and the effect of the injection into the conchae differs more from that of intravenous injections. Although in one case some of the introduced particles were found in the tonsil, they were so few in comparison with the abundance found in the regional lymph nodes that there is no reason for ascribing to the tonsils a local rôle in the drainage of lymph from the nose.

EDITOR'S ABSTRACT.

TREATMENT OF OZENA WITH LACTIC ACID AND ACETYLCHOLINE. H. KOBRAK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:89 (May) 1933.

In the opinion of Kobrak the theory of Guns and Coene that ozena is a trophic disturbance resulting from changes in the sympathetic nerve fibers is not proved

by the success of their combined lactic acid and acetylcholine therapy. In personal experiments he demonstrated that patients who were treated only with lactic acid showed the same signs of improvement as those treated with lactic acid and acetylcholine.

EDITOR'S ABSTRACT.

Miscellaneous

FURTHER STUDIES OF RHINOLALIA APERTA. DAVID L. POE, Arch. Pediat. **50**: 147 (March) 1933.

Attention is called to the occasional occurrence of nasal speech following operations for the removal of tonsils and adenoids and to the persistence of the nasal quality of the voice following closure of a cleft palate. The mechanism of voice production and the disturbing influence of such operations to this mechanism are discussed in some detail. Present day methods of therapy looking toward the correction of nasal speech are discussed, particularly the use of electric current, stretching, massaging and speaking exercises.

WADDELL, JR., University, Va. [AM. J. DIS. CHILD.]

CIRRHOSIS OF LUNG DUE TO ASPIRATION OF LIQUID PETROLATUM. H. BODMER and P. KALLÓS, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **136**:40 (Aug. 24) 1933.

Bodmer and Kallós report a case of Corrigan's chronic cirrhosis of the lung (roentgenologically diagnosed) which they think was due to repeated aspirations of liquid petrolatum over a long period. The patient was in the habit of making daily instillations of from 50 to 100 cc. of liquid petrolatum into his nose because of a pachydermia of the vocal cords and a predisposition to catarrhal attacks. He instilled the liquid slowly into both nostrils, and brought it into the nasopharynx by tilting the head back; he purposely aspirated some oi the petrolatum to oil his vocal cords. The patient had been doing this for ten years. As tuberculosis, syphilis, silicosis and pneumonoconiosis were anamnestically unlikely as causes of the cirrhosis of the lung in this case, the authors attributed the cirrhosis to the foreign body irritation caused by the petrolatum. This assumption is supported by the recent observation of a similar case by Fischer-Wasels, in which from 100 to 120 Gm. of liquid petrolatum was found in the cirrhotic lung at necropsy, the patient having instilled mentholated petrolatum into the nostrils daily for twenty years. Liquid petrolatum is not a saponifiable fat and remains in the place to which it has penetrated, producing a chronic irritation. These observations indicate that it would be wise to examine roentgenologically the lungs of all patients treated with liquid petrolatum for a long period or possibly to use saponifiable fat in place of petrolatum.

EDITOR'S ABSTRACT.

Society Transactions

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

Thirty-Ninth Annual Meeting, Chicago, June 8-10, 1933

JOSEPH C. BECK, M.D., Chicago, *President*

PRESIDENTIAL ADDRESS. DR. JOSEPH C. BECK.

The president in his opening address called attention to the following subjects which appear among those that are, at present, of greatest importance to the otolaryngologist: (1) neoplasms of the nose, throat and ear and borderline regions; (2) allergic disease of the nose and throat; (3) the common cold; (4) pathologic changes in the ears, nose and throat, accompanied by changes in the blood picture; (5) sinus disease in children; (6) mastoid disease; (7) intracranial complications arising from disease of the ear and nose; (8) cause, prevention and treatment of chronic deafness; (9) reconstruction of deformities of the head by means of plastic surgery; (10) influence of educational, social and economic factors on all of the aforementioned conditions.

THE USE OF AMNIOTIC MEMBRANE AS A TRANSPLANT FOLLOWING RADICAL MASTOIDECTOMY. DR. HORACE R. LYONS, Chicago.

The development of radical mastoidectomy was slow and has been modified frequently because the end-result is unsatisfactory in that too often a chronic otorrhea persists after operation. To increase the usefulness of radical mastoidectomy, the author presented the use of amniotic membrane as a transplant to augment epidermization of the radical cavity.

Amniotic membrane is a relatively durable membrane, without blood vessels, and obtains its nourishment by absorption. It is obtainable in considerable size, so that a single piece is used in the radical cavity. The membrane is obtained fresh and preserved in physiologic solution of sodium chloride at body temperature. Necessary precautions include a negative Wassermann test on the donor's blood and a negative Kahn test on the placental blood. The amnion and chorion are separated when the radical cavity is completed and cut to size, and the surface adjacent to the chorion is placed against the bone in the ear. The transplant extends only to the facial ridge, is tucked into the eustachian tube and is packed with gauze saturated with liquid petrolatum. Daily dressings are done, and the pack is saturated with hydrogen dioxide and peeled out carefully on from the ninth to the twelfth day. In the event of copious discharge, a drain is inserted in the lower end of the incision in the mastoid.

The technic of radical mastoidectomy is not discussed except to stress the necessity of a large radical cavity and the elimination of all chronic disease in the middle ear and mastoid.

Two postoperative cases in which the amniotic transplant was used and which had been observed for more than one year were reported.

Comment.—These two cases, each of which was observed for approximately one year, exhibited the following evidence: 1. The immediate postoperative course in the hospital was not complicated in any way as a result of the use of the amniotic transplant. 2. The duration of the postoperative discharge was definitely reduced, especially in the second case. 3. It seems probable that the part of the amniotic membrane that becomes fixed reduces the time otherwise necessary to cover the radical cavity and also reduces the area involved by granulation tissue.

DISCUSSION

DR. GEORGE E. SHAMBAUGH, JR., Chicago: The surgeon who undertakes to carry out the radical operation on the mastoid has before him several rather difficult hurdles. First, and perhaps the most important, is the selection of cases in which this operation is indicated. This depends on the recognition, usually, of a cholesteatoma, and it should be emphasized that the majority of cholesteatomas can be satisfactorily controlled by local measures and do not need the radical operation.

The second hurdle is the operation itself. When the radical operation on the mastoid was first devised, it was the rule in Politzer's clinic in Vienna to require that one should have performed this operation at least fifty times on the cadaver before undertaking it on the living patient. With respect to the technic of operation, I should like to mention one point. As frequently described in textbooks, the first stage of the operation is to do a simple mastoidectomy, leaving intact the posterior and superior canal walls, including the spine of Henle, as a landmark until the second stage is begun. This is a mistake, because in the cases in which radical mastoidectomy is indicated, the disease is not in the mastoid cells but in the attic and antrum of the middle ear. Moreover, in these chronic cases the mastoid is usually sclerotic, and the lateral sinus is far forward and apt to be injured by the usual simple technic. The operation can be expedited and injury to the sinus avoided by taking down the posterior and superior canal walls and the region of Henle's spine first. Once the cholesteatoma has been found, the radical cavity is completed and enlarged to the size necessary to obtain permanent, wide, free drainage.

The third hurdle is the one of caring for the operative wound so as to hasten and insure complete epidermization of the radical cavity. Dr. Lyons has presented an entirely new and original method of surmounting this last hurdle. In attempting to evaluate this contribution, let us first consider the causes of persistent moisture after the radical operation on the mastoid. The most frequent cause is the exuberant growth of granulations leading to pocketing and failure of proper epidermization. The second cause is persistent mucus from the eustachian tube and mucosa of the tympanic cavity. Formerly, considerable care was taken to curet out the eustachian tube, but it has been pointed out that to curet out all of the mucosa from the tympanic cavity is impossible and dangerous. Moreover, slight moisture from the tube or the middle ear is not a source of danger and causes very little inconvenience to the patient. Third, failure to obtain a dry ear occurs occasionally in those cases of cholesteatoma in which the matrix has invaded the small cells of the mastoid around the antrum, or even the haversian canals in the bone, so that the walls of the completed radical cavity are honeycombed by tiny pockets of cholesteatoma which continue to exude moisture. When this type of cholesteatoma is encountered at operation, this invaded bone must be thoroughly curetted out, whereas usually the smooth lining of a cholesteatoma cavity is carefully preserved to hasten healing.

Dr. Lyons has presented an ingenious method of preventing the first and most frequent cause of failure to obtain a dry ear. The question that comes to mind is, what advantage has his procedure over the skin graft? When the paraffined gauze basket mold devised by Mosher is used, a skin graft is easily applied to the walls of the radical cavity.

Amniotic membrane is covered by a single layer of flattened epithelial cells but it is not epidermis. It evidently helps to keep down granulations, but eventually the cavity must be lined by epidermis. Why not use epidermis in the first place?

DR. SAMUEL J. KOPETZKY, New York: I have no desire to discuss the paper for the reason that the two cases reported were not classic ones. In the first place, the first case, as I understand it, was one of a pneumatized mastoid process and did not heal under local treatment. The second case was one of cholesteatoma with polyps. That is a rather rare finding.

In regard to the technic of applying amniotic membrane, I have had no experience. We have recently revived the technic of Dr. Todd of using the cholesteatoma matrix to line the cavity, and we get over all the hurdles that Dr. Shambaugh spoke about. A dry ear is obtainable; there are rapid healing and prevention of granulation tissue. The matrix does those things very well. He asked: "Why not use epidermis?" One does line the cavity with epidermis because there is no difference histologically between cholesteatoma matrix and epidermis.

DR. HORACE R. LYONS, Chicago: I am sorry Dr. Furstenberg is not here. In a telephone conversation this morning he said that he wanted to report that in the laboratory of the University Hospital at Ann Arbor, Mich., the technicians have been cultivating and growing amniotic membrane.

In regard to Dr. Shambaugh's discussion, I think that any transplant or graft used in a cavity must be preceded by a very radical mastoidectomy.

What becomes of the transplant? One of these cases has been followed up for a year and three months, and the other for just over a year. These two cases were reported only because the others are too new on which to base an opinion. However, in the two reported, the transplant formed the base and the epithelium has grown over the transplant, so that as the radical cavity fills in, the transplant disappears.

I realize, Dr. Kopetzky, that two cases do not comprise a very extensive series, but they did show enough evidence to those of us who saw them to make a report worth while. They were reported with the hope that some other members of the Society will use the technic and see whether it adds anything to our general technic.

I believe that some method other than that which is now in use is necessary, because there is too large a percentage of discharge from the ears after radical mastoidectomies, as most of us apply a great majority of these operations to our service patients. Of course, our private patients are better cared for. Certainly, the greater number of radical mastoidectomies are performed on the service and dispensary type of patient.

I do not know what the laboratory is doing at Ann Arbor. Dr. Furstenberg is very much interested in the growth of the amniotic membrane. I should be glad to correspond with any one who would like to try it to see whether it works out with any success.

A RISE IN TEMPERATURE AFTER MASTOIDECTOMY. DR. HENRY P. JOHNSON, Portland, Me.

A study was made of the signficance of the rise in temperature following mastoidectomy in a series of a hundred consecutive cases. The effect of the operation on the temperature curve during the first twenty-four hours was observed in the whole series. It was noted that: (1) There was a drop in the temperature within from six to twelve hours after operation in 66 per cent of the cases, while in 28 per cent there was an elevation; (2) within from twelve to twenty-four hours after operation 86 per cent of the patients had an elevation in temperature, while 8 per cent had a decrease; (3) there were six patients who were afebrile before operation and during convalescence.

There were sixty patients who had an uninterrupted convalescence and were without preoperative complications. The postoperative febrile period lasted from one to thirteen days, the average being five days. The greatest elevation in temperature occurred in 80 per cent of the patients at some time during the first twenty-four hours after operation. It was above 103 F. in only 10 per cent, and did not exceed 101 F. in more than 80 per cent. The fever was not accompanied by other symptoms and had no untoward effects on the patient.

Four patients had preoperative complications of otitic origin, and the postoperative temperature was of no special diagnostic interest. One of these patients died of meningitis on the fifth postoperative day.

Four of the thirty-six cases in which postoperative complications developed were of a serious nature. There were three patients with an involvement of the lateral sinus who recovered following surgical intervention. One patient died on the fourth postoperative day of general sepsis; the cause for systemic invasion was not determined. An elevation of the temperature curve was the first warning of an existing complication in more than 86 per cent of the cases.

DISCUSSION

DR. H. I. LILLIE, Rochester, Minn.: Reports of statistical studies of series of cases give valuable information and are extremely useful to the clinician in his daily practice. They establish precedents and may prevent clinicians from becoming panicky when some of their cases do not follow certain described clinical courses.

Dr. Johnson has shown that the importance of fever early in the postoperative course is at times difficult to evaluate. It may be a favorable sign if it suggests that the patient is resisting the effect of the infection, or it may be an unfavorable sign if it suggests that the patient has been overwhelmed. Only when taken into consideration along with findings resulting from careful examinations can the real significance be estimated. Causes of fever other than the infection in the ear may be present.

A gradual decrease in temperature following operation is looked on as a favorable prognostic sign. Any sudden rise in temperature following a relatively uneventful postoperative course is most significant and demands close observation to determine the cause.

Certain persons seem to have a tendency to fever from insignificant causes, whereas others maintain a normal temperature under the same circumstances. A physician's clinical judgment is dependent on his ability to interpret the significance of the composite clinical picture.

DR. STANWOOD E. FISHER, Portland, Me.: Fever of some degree of elevation is practically always present in every case of infection of the mastoid both before and for a while after operation. The vast majority of cases are of this type. Each may be a little different from the next one, but the general run of uncomplicated cases is the same. I shall emphasize the fact that the temperature is the first thing on the chart that one looks for; it tells at a glance whether or not the case is progressing as it should. It is indeed rare to have a patient do badly in whom elevation in temperature is not the first response. It is the red flag that nature puts out, if things are not as they should be.

The sudden rise in temperature immediately following operation, often seen in children, is of no serious moment and should cause no concern, for within twenty-four hours there will be a rapid drop followed by a nearly normal temperature. It is more apt to be seen in early operations, before the protective wall is formed. After one week of convalescence, if there is a sudden elevation in temperature, this is generally the first symptom of some complication, and a thorough search should be made to find the exact cause of the elevation.

I cannot emphasize too strongly the importance of regular and frequent taking of the temperature during the period of hospitalization in all cases of infection of the mastoid.

DR. LYMAN G. RICHARDS, Boston: I had the opportunity to read Dr. Johnson's paper, following which I took the pains to look up a large number of cases of mastoiditis at the Children's Hospital in Boston.

I should like to present the results of the summation of these cases. The lower curve on the slide shows the average febrile reaction in a hundred cases of simple unilateral mastoiditis, uncomplicated by any essential pathologic conditions. You will notice that the preoperative temperature starts a little above 100 F. As Dr. Johnson showed, there is a slight reaction on the day of operation and on

the next day. Subsequently there is a slow and practically continuous and satisfactory convalescence throughout a period of two weeks, which is the period during which we collected these cases.

It is interesting to note that there was practically no difference between a group of forty-eight private cases (in which, by the way, operation was performed by a large group of men in the hospital and not always by me) and a group of fifty-two cases in the wards. In spite of the fact that cases in a ward are assumed to be less favorable for operation and more apt to run a complicated course, there was no difference in the postoperative reaction of the two groups. From that fact it is fair to assume that in a simple case of mastoiditis one may normally expect an essentially uncomplicated course.

If, during that period, particularly the latter part, there is a febrile reaction, one can no longer attribute it to a so-called postoperative reaction or to any of the other factors which may have been blamed at certain times. Therefore, the rise must in some way be explained.

The relatively minor complications, although they do not show on this chart, come in about this period; they comprise cervical adenitis, development of complications in the other ear and all the things that Dr. Johnson mentioned. I think that it is safe to allow a certain amount of time to pass before one becomes excited about these complications, and to assume that the later they appear the less likely they are to be serious.

In contrast to this, we collected a smaller, though sufficient, number of cases of sinus thrombosis. I know that before I got this group together I had the impression, which I think one gains from textbooks, that sinus thrombosis is apt to pursue a course similar to that of uncomplicated mastoiditis for from eight to twelve days, and then suddenly creates complications. I was very much surprised to find certain features in connection with this group of cases of sinus thrombosis. The patients all had a higher preoperative temperature to begin with, before one knew that sinus thrombosis was developing. The average was 103 F., as contrasted with 100 F. in the simple cases. Following the operation, the condition ran a more or less irregular course. In other words, from the very beginning one suspected that there was something wrong, that the patients were not doing well. Although sinus thrombosis may not be evident, the cases must be kept under observation, with the idea that sinus thrombosis may subsequently develop. In many cases operation may be safely delayed until a diagnosis can be established.

The question of the delay in development of sinus thrombosis is another matter, and there is no time to go into it. In certain cases in which the febrile reaction arises at a later period, one must decide whether the condition is thrombosis and must not mistake it for erysipelas or pyelitis.

The third group comprises cases in which meningitis developed. There was a higher febrile reaction in those cases than in any of the others. Without the fluctuation that occurred in the cases of sinus thrombosis, the high fever persisted throughout the course. As you know, most of the cases resulted fatally.

The operative procedures are not indicated on the chart. They were performed at various intervals, when the operator thought that something should be done.

The point I wish to leave with you is that the more serious type of cases will at the outset show evidence of a higher and more severe febrile reaction, and if the reaction is maintained almost from the beginning—it may possibly quiet down, as it has in some of the cases observed—it is an indication that trouble lies ahead. One must be more watchful of that type of case than of those in which the febrile reaction develops at a later date.

Dr. J. W. Jervey, Greenville, S. C.: Dr. Johnson has given a very illuminating and highly practical discussion, and Dr. Richards has further illuminated it. In this connection, I should like to mention briefly a rather interesting situation. A boy of 9 years was operated on for simple mastoiditis. No preoperative complications were suspected. However, following the operation

every afternoon for five or six days the temperature rose to 105 F. At that time an infection of the lateral sinus was suspected, and a blood culture was made. By the time we received the report on the blood culture, the wound in the mastoid and the ear were clean and dry; however, the report was positive for Bacillus typhosus. At the time of operation the smear culture showed streptococci. No further investigation was made as to any complication or additional infection. On the sixth or seventh day, an acute infection of the mastoid developed in the other ear. It seems to me that I have read of very few cases of mastoiditis supposedly due to Bacillus typhosus, and, so far as I know, that organism is not stressed as a causative organism.

DR. HENRY P. JOHNSON, Portland, Me.: In the study of this group of unselected cases, an attempt has been made to interpret the fever following mastoidectomy. It was found that the fever following operation ordinarily should not be a source of concern to the surgeon unless there are other symptoms accompanying it. The elevation in temperature which occurred in the first twelve to twenty-four hours was to be expected. It is usually of no special significance and should not be a source of anxiety. However, when a satisfactory explanation cannot be given for a fever which occurs after the first three or four days, it should be considered serious, and a search should be begun at once for other symptoms to determine the cause. A rise in temperature was the first warning of approaching trouble in the majority of complicated cases in this group.

THE REFLEX CONTRACTIONS OF THE MUSCLES OF THE MIDDLE EAR AS A HEARING TEST IN EXPERIMENTAL ANIMALS. DR. RAFAEL LORENTE DE NÓ, St. Louis.

Experimental work has been done on the rabbit in which the tensor tympani muscle has been used as an indicator of perception of hearing. There exists a reflex arc in which stimulation coming from the auditory perception apparatus has a twofold effect: (1) on the cortical center of perception of hearing and (2) on the tensor tympani muscle, demonstrated by apparatus whereby the muscle is connected with a light-reflecting apparatus translated as a curve on a suitable chart, the effect of stimulation of the auditory apparatus being recorded in terms of response of the tensor tympani muscle. This response has been found to parallel closely that obtained in testing hearing in human beings and likewise to be influenced by similar factors. Thus, for instance, perforation of the drum elevated the curve in both the human being and the rabbit. No tympanic reflex, however, can be elicited below 128 double vibrations.

DISCUSSION

DR. S. POLJAK, Chicago: After having heard the fine paper by Dr. Lorente de Nó and after having read several of his previous papers dealing with the structure and the function of the cochlea and the primary nuclei, I believe that here again we have a real step forward in an understanding of the whole auditory system.

Perhaps it would be well to say a few words about my own work. About eight years ago I became a little more interested in the auditory system, and I was struck by the simplest facts about the acoustic system. It is natural that the theorists who were trying to explain the function of the auditory system largely disregarded the acoustic system, especially the nerve part, the nerve apparatus of the acoustic system. They paid attention mainly to the cochlea itself and especially to the physical features of the cochlea, but how the nerve apparatus participates in this function and what is the basis of this function were largely unknown.

Now since we know from Dr. Lorente de Nó's work that there is at any rate a kind of projection of the cochlea on the central nervous system, we come a little closer to understanding it.

In order to explain what I mean, I should like to give as an example another system, that is, the visual system, in the understanding of which we have made a much greater advance. You know, perhaps, that since the beginning of the study of the motor area, the study of the brain and so on, there was always the question connected with the visual system and the nervous system: Is there a projection of the peripheral receptor of the retina on the nervous system, especially on the brain, which means a projection that would throw or produce a representation of parts or quadrants of the retina on certain parts of the cerebral cortex? This has been accepted by some people and denied by others. Nevertheless, recently, in the last ten or fifteen years, we have collected sufficient data so that at present we accept the fact that there is a very fine projection of the retina on the cortex. I shall take the liberty of showing a few of my slides which deal with this particular question.

DR. J. GORDON WILSON, Chicago: Dr. Lorente de Nó's paper is of great importance, not only because of the findings and interpretation, but chiefly because he has uncovered a new method of approach to the study of auditory function. He enters this investigation equipped with a knowledge of the cochlear nerve and its intracranial ramifications beyond that possessed by most investigators in the field of electrical response to auditory stimuli. A striking example of this is his explanation of the relationship of the nuclei of the fifth and seventh nerves and the superior olive, which brings into association the nerve supply of the stapedius and that of the tensor tympani muscle and links each with the auditory pathway in the olive.

Dr. Lorente de Nó has presented the results of his observations on reflex contraction of the muscles of the middle ear to sound which he hopes will lead to a test of auditory acuity in experimental animals.

There has long been desired, but not yet found, an auditory reflex, involuntary and constant, comparable to the involuntary reflex of the muscles of the eye which has helped so much to an understanding of the function of the labyrinth. With such a reflex one might hope for light on some of the auditory problems which are baffling us today. Within the last few years there has come a great revival of interest in the nerve mechanism of audition, together with new methods of approach. There has been more intense study of conditioned animals and of the phenomena associated with the Wever-Bray experiment, and now the reflex contraction of the muscles of the ear which Dr. Lorente de Nó has studied.

I bid you note that the function of the muscles of the ear is little known, least of all in man. Are the muscles protective against loud sounds? Do they act as muscles of accommodation? Is the tensor tympani a tensor tympani or a misnomer? Have they any function in man (though they may have in the lower mammals)?

Luckily the tensor muscle in animals is a more massive muscle than in man, and as Dr. Lorente de Nó has demonstrated, the tensor muscle responds to sound vibration.

The study of the effect of sound impulses entering the ear with an intact cochlea can be carried out in three ways: by the Wever-Bray method, by Dr. Lorente de Nó's method and on conditioned animals.

In the Wever-Bray effect during the passage of sound there is an electrical pick-up of variations in potential in the auditory nerve pathway. This has given many interesting and curious results, but the interpretation of these results and their relation to hearing have caused much controversy. The currents picked up from the auditory pathway between the cochlea and the cortex have frequencies, like the frequency of action currents in sensory nerves elsewhere, not higher than about 900 per second and so do not suggest that they will help much in the study of pitches or vibrations that go far over 1,000. But associated with these action currents there can be picked up a second phenomenon (the spread phenomenon), studied especially by Davis and his co-workers at Harvard, not confined to the auditory nerve pathway but spread diffusely through the tissues of the brain. They believe that it is to this spread phenomenon that the higher frequencies and intelligibility of speech are due; this intelligibility is the striking result of the Wever-Bray phenomenon. There is no proof, however, that this can be called an

acoustic response. Adrian, the great authority on electric phenomena in nerves, believes that it is due to microphonic action in the cochlea, and that it is an electrical rather than a physiologic manifestation.

What appears to be a more intimate reflex phenomenon is that presented by Dr. Lorente de Nó, who has given an excellent demonstration of these contractions and how they can be recorded. He has shown how the audiogram of reflex contraction bears a resemblance to the audiogram of hearing in man, and his results in removal of the drum membrane fit in well with our experience in clinical otology. He disregards the physiologic function of the muscle and concentrates on the response to sound vibrations. As he is dealing with experimental animals, he can disregard the denunciations of those who believe that the muscles of the middle ear are nonfunctioning in man; but, of course, this will have to be considered if one seeks to apply the findings to man. He has demonstrated that he can get a uniform reflex muscular response varying quantitatively with the intensity of the acoustic stimulus, which at all times has to be some distance above the threshold. This is in keeping with known reflex responses to stimuli in other muscles. But in the uniform reflex he points out there is a special feature which he believes is the expression of a qualitative difference due to pitch. This explanation, if confirmed, is a fact of great importance.

But there are difficulties still to be overcome and discrepancies to be explained. Hughson and Crowe (Experimental Investigation of the Physiology of the Ear, *Acta oto-laryng.* **18**:309, 1933) made repeated attempts to determine whether visible contraction of the tensor tympani muscle took place as a result of sound stimuli. Observing the muscle under the microscope and using the lowest pitched fork (32 double vibrations) as a stimulus, or the lowest tone on the oscillator scale, it was never possible to observe the slightest evidence of contraction. This is in line with Kato's results, who noted only reaction to sound *am freigelegten Muskel* (Mangold, Ernst: Handbuch der normalen und pathologischen Physiologie, vol. 1, pt. 1, p. 428). The Baltimore investigators also found that when the tensor is divided and visible, contraction of the muscle takes place when the pinna is pinched or the external canal stimulated. Though the tensor is innervated by a branch of the fifth nerve, direct stimulation of the facial nerve or any of its branches caused a sharp contraction of the tensor muscle and also mechanical irritation of the chorda tympani.

I find it hard to believe that in the reflex which Dr. Lorente de Nó demonstrated we are dealing directly with an auditory phenomenon; the pathways are remote, and there are too many extraneous factors. Nor does Dr. Lorente de Nó claim it to be so. I believe, however, that the findings presented show that there is some relationship. May I suggest that here we may be dealing with a function of auditory acuity, that the two responses are related so that a variation in one produces a variation in the other? It remains to be seen whether they are sufficiently clear and constant to be of use.

The third method that has been used in the study of audition is the action of sound on the cerebral cortex. It is with this that I have been most in contact. I believe it offers the best approach to an understanding of auditory function as a whole. For instance, the animal is not anesthetized, and therefore is more normal. It offers a means of separating pure tone perception and therefore hearing from vibrations (mechanical) so apt to complicate tone production—these are two independent entities with two separate cranial associations. The results so far obtained have been promising.

All three methods of approach are working toward a common goal. So far all have disadvantages, but each in its own field will give something to help explain unknown problems in hearing. In conclusion, let me point out that Dr. Lorente de Nó is blazing a new pathway, and no one expects, no one has the right to expect, that the signs along the way are as legible today as they will become when the path has been trodden by many feet over many years.

DR. RAFAEL LORENTE DE NÓ, St. Louis: I wish to say a few words of thanks to Dr. Poljak and Dr. Gordon Wilson for their thorough discussion. The

work of Dr. Poljak on the projection of the optical system is extremely interesting. In regard to the acoustic pathway, I hope he will carry out a similar investigation and give us a clear concept of what happens with the last neuron.

I believe that the projection in the cochlear system will at the end be a little bit different or according to a different principle than the projection in the optical system.

To Dr. Wilson I shall make a few remarks concerning the fact that the tensor tympani contracts on the touching of the skin; the last myogram that I projected was obtained by excitation of the auricular nerve. I have been using this sensory difference in order to test the motor neurons of the same properties as the other motor neurons. This shows that when the tone is produced, the reflex is not purely acoustic. The pupil in the eye contracts not only when light falls on the eye, but also when pain is produced. The fact that the pupil contracts when pain is produced does not show that the pupillary reflex is not an optical one. Contraction can be produced in every muscle through many different ways. The tensor tympani can be brought to contraction in many different ways. If one takes care that the stimulus is purely acoustic, one will obtain a pure acoustic reflex. I have been basing all the studies on the acoustic reflex.

The tensor tympani reflex, as the audiogram shows, appears only with tones over 128 cycles. With tones lower than that, the reflex never appears. The function of the muscle is the point that future research will show. At present I do not disregard the function of the muscle, but I think that we know entirely too little to establish a theory about the function of the muscle.

THE SURGICAL IMPORTANCE OF THE MASTOID VEIN IN INFECTED LATERAL SINUS THROMBOSIS. DR. EDWARD F. ZIEGELMAN, San Francisco.

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DISCUSSION

DR. H. I. LILLIE, Rochester, Minn.: Dr. Ziegelman has presented a subject that is of anatomic interest and of great clinical significance. Three years ago, a case was encountered in which a thrombus was found in the emissary vein. The importance of extending surgical intervention until tissue is encountered which has undergone sufficient pathologic change to account for the clinical course was well borne out. In this particular case the symptoms were recognized early, and at operation hemorrhagic mastoiditis was encountered. The sinus plate appeared normal, as did the wall of the sigmoid sinus when the plate was purposefully removed. Exposure was extended posteriorly to the lateral sinus, and here phlebitis was encountered where the emissary vein emptied into the sinus. The vein emptied into the sinus on its outer surface. It could be seen that there was a definite thrombus within the vein. The thrombosed portion of the vein was removed, and the lateral sinus was obliterated. The patient recovered.

It has always been my understanding that the emissary vein empties into the lateral sinus. I know from clinical experience that it varies in size and in the point at which it empties into the sinus. It is difficult to see how the external jugular vein could be involved in the disease of the mastoid if the structures were of the usual anatomic development. Anatomists mention the fact that the occipital vein and the posterior auricular vein may empty into the internal jugular vein. In a mastoid process in which there was an extensive cellular arrangement behind the sigmoid sinus, it is easy to see how the emissary vein could be involved by disease. It would seem likely that the condition would be revealed at the time of operation on the mastoid. Unusual or anomalous conditions of the common facial vein are not uncommon. I have observed thrombosis of the common facial vein extending to the tributary that goes to form the external jugular vein. It is easy to see how extension of such thrombosis could involve the external jugular vein.

It is well to know of these unusual conditions because they may be encountered at any time. Familiarity with the anomalies of the vascular system is an important part of the armamentarium of the otologist.

DR. SAMUEL J. KOPETZKY, New York: This is a very important topic as a side issue to the management of lateral sinus thrombosis. My attention was first drawn to the emissary vein of the mastoid in tracing the egress of pus that formed a subperiosteal abscess. In a certain number of cases the leakage occurred through the foramen through which the vein came. In a few instances the vein itself was involved. In one instance in my records there was a distinct thrombosis of the emissary vein of the mastoid, not, however, extending to any large area of its length.

Thrombosis occurs both against and with the blood stream, so the natural anatomic area in which the vein drains is of less importance because the thrombus will grow both with and against the stream.

Through carelessness in retracting the soft parts during the primary operation on the mastoid process, very often the emissary vein is injured, not necessarily at its point of emergence through the mastoid but in the soft tissues as they are roughly retracted. Thereafter there develop infection and tumefaction of the tissue. It is then well, in accounting for the sepsis, when one inspects the wound and examines the sinus proper and finds everything in order, to look in that area for trouble, the surgeon being partly responsible for it. Primary thrombosis can and does occur and should be borne in mind.

With the excellent paper before us and the discussion of Dr. Lillie there comes to mind the management of the spread of infection in this area, when dealing with cases of thrombosis of the lateral sinus. One tries, naturally enough, to remove the thrombus in all accessible areas within the lateral sinus. In doing so it is feasible also to block off the emissary vein, just as one blocks off the petrosal vein if one has any suspicion of phlebitis, primary to an extension of the thrombosis into that vein.

DR. ROBERT SONNENSCHEIN, Chicago: With reference to a primary thrombosis of the emissary vein, let me report very briefly the case of a colleague in which, following acute otitis media, there were all the clinical signs and symptoms of lateral sinus thrombosis, namely, the chills, the peaklike temperature and other symptoms.

At operation the sinus appeared normal. However, after blocking and opening it and finding blood in it, we saw that a thrombus from the emissary vein was projecting into the lumen of the sinus, that there was no thrombosis in the sinus itself. It was a case of very fulminating septicemia, and, unfortunately, death occurred.

DR. EDWARD F. ZIEGELMAN, San Francisco: Dr. Kopetzky brought out a very interesting point, the blocking off of the emissary vein at the time of operation. I think that that is a point we should all remember. There is very little for me to add.

THE SPREAD AND EXTENSION OF PARTICULATE MATTER IN THE NASAL MUCOUS MEMBRANE OF THE RABBIT. DR. BERNARD J. MCMAHON, St. Louis.

The changes in the tissue of the nasal mucous membrane in the presence of infection have repeatedly been subjected to experimental study. The chief reactions have been found to be in the connective tissue elements rather than in the epithelial or subepithelial layers. A question of regeneration of nasal mucous membrane has been thoroughly worked out, and the fluid changes on the surface of the mucosa have likewise been studied. Of late much attention has been given to the histologic details of the connective tissue layer and to the cells such as histiocytes produced by vascular epithelium. The present work was undertaken (1) to see whether particulate matter such as india ink would penetrate normal nasal mucosa and (2) to see the reaction if this material was injected into the subepithelial tissues.

In the first experiment the diluted ink was sprayed into the nose for a contact of thirty minutes, the animal killed in six hours and the turbinate removed, fixed in Zenker's solution and embedded for serial section. In the second case diluted ink was injected into the antrum, and the animal similarly killed. In the third experiment the ink was injected into the anterior tip of the inferior turbinate. From these experiments it was found that the ink does not penetrate into the epithelium in the nose or the antrum. In the antrum the ink is phagocytosed by histiocytes and not by polymorphonuclear leukocytes, the latter being more prone to ingest bacteria and to be themselves subsequently phagocytosed by histiocytes. In about twenty-four hours there was found to be an occlusion of lymphatics, the particles traveling through the lymphatics into the tissues. One can thus conclude that lymphatic block may spread infection.

DISCUSSION

DR. WILLIAM V. MULLIN, Cleveland: Let us hope that Dr. McMahon will go on with his valuable investigation. As he truthfully states, there is still much to be done. He has not added much to what Fenton has already told us about the histiocyte. He has referred to the work I did on the sinuses with india ink. I think that if he reviews the original reports, he will find that they embody nearly all the points brought out in his paper.

I quote a most essential portion from Dr. McMahon's paper and what would seem to me to be the thing most necessary to investigate further.

"The lack of penetrability of inert matter and the absence of any attraction for it by the epithelium in toto, or by its cells individually, would imply the necessity for an invading body to be biologically, electrically or osmotically active in order to create a condition of mutual attraction with the epithelial cells of the nasal and antral mucosa."

Most injured tissues become acid, and the potential is changed. Dr. Maria Telkes in the biophysics laboratory of the Cleveland Clinic Foundation has carried on some experiments, measuring the potential difference of single cells like the ameba. The amebas in their normal surroundings have a definite negative charge. This fact has been verified by others. If various physiologic agents are added to the culture medium, the potential difference of the ameba changes. Distilled water immediately increased the potential difference.

If the physiologic agent is a very dilute solution of sodium chloride, the potential changes scarcely at all; if the solution is more concentrated, the potential difference diminishes, in a few minutes returning to normal. If, however, the salt solution is even more concentrated, the potential difference drops permanently to zero, and the ameba dies. This same phenomenon was observed with a great number of physiologic agents, for example, magnesium chloride, calcium chloride, phosphates and even physiologic poisons like morphine and strychnine, and also with epinephrine, iodides and other drugs.

As a conclusion it was found that the physiologic agents changed the potential difference of the ameba, and that the ameba's viability is proportional to its electric charge. To prove this further, an electrode was introduced into the ameba, and an extraneous electromotive force was applied to the ameba. If this force was counteracting the ameba's own charge, the ameba died in a few minutes. If the charge was in the same direction as the ameba's own charge, the ameba became more motile. Too strong a charge applied in either direction killed the ameba.

It may be possible to apply these findings in further studies of the sort Dr. McMahon has presented, and to glean therefrom more definite information as to the relative importance of the various factors involved in changes in the nasal and antral mucosa.

DR. RALPH A. FENTON, Portland, Ore.: Dr. McMahon's findings abundantly corroborate our work on the mucosa of the cat's frontal sinus, in that no phagocytosis by epithelial cells is observed, and also that phagocytosis in the subepithelial layer occurs mainly in the connective tissue cells—histiocytes especially

—and not in polymorphonuclears. In tissues subjected to trauma, heat and chemical stimuli, we have demonstrated that in the absence of definite infection these connective tissue cells seem to be mobilized earlier than are the polymorphonuclears. The presence of polymorphonuclears in the lumen of a sinus would seem to indicate a coincidental infective process, thus somewhat vitiating the results of injection of sterile inert matter such as finely divided carbon.

In tissue cultures of nasal mucosa stained by Dr. Ross with neutral red, in Dr. Mosher's laboratory, one may see histiocytes full of red granules engaged in active pseudopodial advance toward traumatized areas, elbowing their way among the epithelial cells, the cilia of which keep on waving regularly; and in this living tissue very few polymorphonuclears are visible. We believe that histiocytes, being at all times latent, ordinarily inactive components of the submucosa, are therefore likely to be put on the defensive in time of need before the polymorphonuclears arrive. Undoubtedly the histiocytes take care of all sorts of débris, including broken-down polymorphonuclears and their contents, which may be more or less toxic; we have, further, demonstrated the mobilization of histiocytes in profusion at the site of trauma and in response to chemical stimulation, with little or no polymorphonuclear infiltration.

Fibroblasts do not appear ever to be transformed into ameboid wandering cells; hence it is doubtful that they have phagocytic powers, as fibroblasts. Since histiocytes are sometimes transformed into fibroblasts, the possibility is raised that the carbon-containing fibroblasts observed may be transformed histiocytes, which had picked up the carbon as histiocytes, but had subsequently become converted into the rather specialized, nonreversible fibroblasts. Possibly irritative conditions set up by carbon particles may have hastened and provoked such metamorpohsis. This might have an interesting bearing on the whole problem of mucosal irritation and fibrosis.

It should be emphasized that the power of phagocytosis by connective tissue cells is essentially a defensive factor, rather than a factor in the dissemination of disease by killed cells, because such cells are singularly potent in walling off and disposing of infection, as evidenced long ago by the peritoneal and pleural studies of Gay, Clark, Linton and others. Lymphatic spread, however, encounters no such barriers, for, as Kelemen has demonstrated on cultures of tonsil tissue, there seems to be distinct antipathy between lymphocytes and histiocytes, and none of the latter exist in lymph follicles—they are found only in the capsule or in the trabeculae of such organs.

The point regarding leukocytes and bacterial ingestion in the sinus cavity is interesting and biologically most significant. Instead of washing it out, except to relieve pressure, should not the attack be somehow made from within the body? Let the leukocytes fulfil their biologic function; help the histiocytes to mobilize if the epithelium is broken; find out the necessary chemical and physiologic constitution of the mucosa which makes it resistant, and then seek means to produce this constitution. This may require metabolic changes involving vitamins and the alkaline-earth and halogen content of the blood and the tissue fluids. Dr. McMahon's work carries us far along this difficult road.

OSTEITIS DEFORMANS OF THE BONES OF THE FACE. DR. GORDON B. NEW, Rochester, Minn.

Osteitis deformans is a chronic inflammatory disease, which generally affects the long bones, the bones of the pelvis, the spinal column and the cranium, but which seldom affects the bones of the face. Five cases of this disease with involvement of the bones of the face, which was not of the leontiasis ossea type, were reported.

The disease most often afflicts patients in later life, and is characterized by destruction of bone and formation of bone in different planes, with enlargement of the part.

The etiology, symptoms and roentgen findings and the differential diagnosis, including hyperparathyroidism, leontiasis ossea, osteofibroma, giant cell tumor and

multiple myeloma, were discussed. In most cases there is associated involvement of other bones of the body, and while little can be done to stop the progress of the disease, removal of portions of the bones producing deformities about the face is well worth while.

DISCUSSION

DR. JOHN J. SHEA, Memphis, Tenn.: Dr. New has presented one of his typical monographs, which will remain as a reference on osteitis deformans affecting the bones of the face. The rarity of the disease limits one's personal experience with it, but a theoretical knowledge is necessary for a differential diagnosis from acromegaly.

Osteitis deformans occurs in the latter half of life, and though it was thought to be inflammatory by Paget, it is today considered to be of endocrine origin. Thymus extract was administered to a series of patient with arthritis deformans with the hope that the thymus might prevent the senile changes in the bones. At first we thought that we had acquired a logical remedy, but a more careful study of the cases convinced us that the thymus extract was of little value.

I have reviewed the case histories and the roentgenograms in Dr. Willis C. Campbell's Orthopedic Hospital at Memphis, Tenn., which is the largest privately owned orthopedic hospital in the country. I could find only three instances of osteitis deformans in which the physical examination justified a study of the head. In the most severe of the three cases there were definite bony changes throughout the skull, with a bulging of the anterior wall of the frontal sinus; in the second case there was less protrusion of the forehead; the third case presented the features of acromegaly.

Dr. New's conclusion, "that operative removal for cosmetic purposes is well worth while," is sound advice.

DR. THOMAS E. CARMODY, Denver: Dr. New's presentation has been so thorough that there is no necessity of adding anything to it; besides, I cannot, as my experience with this disease has consisted of only one case that possibly could be diagnosed as osteitis deformans.

From the standpoint of diagnosis, we must take into consideration the cystic tumors of the face, the fibromas of the face, the osteomas of the face and jaws and also inflammatory conditions, as well as sarcomas.

DR. LOUIS K. GUGGENHEIM, St. Louis: I cannot resist the temptation, in conjunction with this brilliant presentation of Dr. New, of saying a word about the similarity between this process and that of osteosclerosis.

Fabre of San Francisco called attention to the fact that osteodystrophica fibrosa, Recklinghausen's disease and Paget's disease are almost identical with the processes of the bone which are seen in otosclerosis, except for the fact that the mosaic which is seen in osteodystrophica fibrosa is absent. It is generally believed that these processes of the bone are due to endocrine disorders, and it has been proved that Paget's disease results from the parathyroid tumor with an excess of parathyroid secretion.

In otosclerosis there is a most interesting limitation of the change in the bone to the densest and one of the smallest bones in the body. Although Fabre believes that the underlying causes in osteodystrophica fibrosa and otosclerosis are identical, we do not agree with him, because in otosclerosis the condition is confined to one part of the body, to a very small part, and no one has ever demonstrated changes in the bones elsewhere.

In otosclerosis there is a hyperostotic and neurotic change as there is in Recklinghausen's disease. There is resorption of the original bone, a replacement with an inferior fiber bone, which is present in all cases. The lamellar bone that is destroyed is never replaced.

I merely wished to call attention to the fact that the processes in the bones in both conditions, otosclerosis as well as generalized changes in the bones, are very similar.

DR. WILLIAM V. MULLIN, Cleveland: I should like to ask Dr. New whether he measured the calcium and phosphates in these cases and whether he found deficiencies or changes.

In one case there was a very considerable deformity around the thorax and the chest. Every time the young man exerted himself he produced some kind of a fracture. For instance, several times in merely putting on his coat he had a fracture. I think that in a period of about nine or ten months he had about a dozen fractures.

I should like to have Dr. Guggenheim tell us more fully about the similarity between otosclerosis and Paget's disease. Some of you may recall that about a year ago Dr. Norval Pierce sent a postcard asking that the hearing be measured and particular attention be paid in cases of Paget's disease. I measured the hearing in fifteen cases of Paget's disease, and in only one was there any symptomatic reference to the hearing; there was no change in hearing. In that case, there happened to be a coincidental suppurative process.

DR. GORDON B. NEW, Rochester, Minn.: The question of calcium deficiency pertains to the hyperparathyroid group. The problem has been worked out on animals. Dr. Wilder and his co-workers studied the question at the University of Chicago on rats. It was excellently worked out and the results were found to be true clinically. So far as I know, calcium deficiency does not occur in osteitis deformans.

A CLINICAL STUDY OF SEVERE DEAFNESS IN ADULTS. DR. GEORGE E. SHAMBAUGH and DR. GEORGE E. SHAMBAUGH, JR., Chicago.

Dr. Shambaugh read a paper, illustrated by numerous audiograms, showing certain characteristic features of deafness in adults.

DISCUSSION

DR. E. W. HAGENS: I believe that this study by Dr. Shambaugh and his co-workers is a step in the right direction. In the leagues for the hard of hearing are necessarily collected those adults whose deafness has become so severe as to become a definite handicap. Therefore, the results obtained in these cases should give a good index as to the probable causes of severe deafness in adults. In considering the total number of severely deaf adults, we may classify them into several groups. First, there are those who were born deaf or who became deaf during childhood and have grown up and are now a part of the adult deaf population. Another group were normal persons until toxic neuritis, labyrinthitis or trauma destroyed their hearing. There are also those in whom the deafness of old age has developed. A small group has deafness which has come on following one or more attacks of infection of the middle ear. A much larger group comprises those whose hearing has become progressively worse without a history of infection of the ear.

Judging from experience with a small group of cases from the night schools for the adult deaf and from private and dispensary practice, I believe that the proportion of cases in the various groups as found by the essayists is essentially correct. I agree with the statement that chronic catarrhal otitis media, per se, is not progressive. Of course, an adult may have repeated attacks of otitis media, each adding its insult, until severe deafness results. Also chronic suppurative otitis media of the bone-invading or cholesteatoma type may progressively invade the ear. However, chronic catarrhal or adhesive otitis media should not of itself produce progressive deafness.

There is one point which I believe should be stressed, that is, microscopic examination of the temporal bones. The value of clinical examination and diagnosis in the cases under discussion would be much enhanced by postmortem study. If a sufficiently large number of cases is studied, the clinical findings will be either confirmed or denied. Perhaps the microscopic results will lead to a new or modified classification of deafness, based on a better understanding of the pathologic changes involved. These things suggest themselves when slides from various cases are

studied. Otosclerosis is found alone and associated with acute otitis media or chronic otitis media or with both acute and chronic otitis media. The diagnosis of otosclerosis clinically is sometimes very positive, sometimes moderately so and at other times somewhat doubtful. If the condition of the ears could be followed through and postmortem examination could be made, we might learn how accurate or inaccurate the clinical diagnosis has been. The foci of otosclerosis are definite under the microscope, and the diagnosis is therefore positive. It is also difficult at times clinically to explain certain combinations of reactions to the functional tests of hearing. I believe that postmortem examination would unravel some of these problems at least. For example, partial deafness of the inner ear localized to a part of the tone scale can be explained under the microscope by finding a localized destruction of the organ of Corti. It seems apparent from the study of the pathologic process of otosclerosis that the treatment in any case sufficiently well developed to be diagnosed clinically would be of no benefit. It would hardly be expected that a thickened stapedial foot plate with obliteration of the normal joint could be restored to normal by any treatment. Prevention of the condition seems to be the only way in which this distressing malady will be overcome.

It is my belief, judging from a study of microscopic sections of temporal bones showing otosclerosis, that this condition is more common than has been supposed. The study of identical twins is a valuable suggestion, yet probably such opportunities will arise but infrequently.

Dr. Robert Sonnenschein, Chicago: This excellent study of a hundred unselected cases from among the members of the Chicago League for the Hard of Hearing forms a nice companion to the more extensive study of severe deafness in children produced by Dr. Shambaugh and several co-workers some years ago under the auspices of the National Research Council. The evidence of the meticulous care with which the patients were examined is most commendable, and the conclusions which were reached seem to me most important. I have long been of the opinion that the very marked impairment of hearing in adults could rarely have been prevented by the periodic examination of school children.

However, do not let me be misunderstood in regard to the importance of the careful examination of the hearing of school children. Certainly it is of greatest importance to determine the various degrees of impairment of hearing by testing children of school age, first of all, in order that proper measures may be taken in those cases in which surgical and other procedures may be of avail. The presence of adenoids or pus in the nasal accessory sinuses and their potential danger to the ears from infection of, or obstruction to, the nasopharynx and the eustachian tubes certainly require intervention. Treatment of suppurative otitis media is important from the standpoint not only of further impairment of hearing, but of the danger of intracranial complications. When dry perforations are present, advice should be given against the entrance of cold water and other substances into the ear, and in chronic suppurative processes, operation may have to be recommended.

Second, and aside from these matters, a careful examination of the hearing of school children is necessary in order that those with a marked degree of impairment of hearing may be segregated from the others; they may be placed in certain portions of the room where they can be nearer the teacher, or they may be placed in groups so that special attention may be given to them, with reference not only to the usual school routine but also to instruction in lip reading. In some instances, when a very marked or almost total loss of hearing is present, children in whom this is noted may be sent to special schools where there are classes either for those with congenital or acquired deafness, or for those who have extremely advanced loss of hearing. Immeasurable benefit would thus accrue to these children to the extent that they would be better fitted for life's work.

However, so far as the prevention of future impairment of hearing is concerned, with the exception of those cases in which there are pathologic conditions previously noted, such as adenoids, lymphoid tissue in the nasopharynx or the

posterior pharyngeal wall, chronic tonsillitis or suppuration of the nasal accessory sinuses, there is very little to be done except the mere examination and classification of the children. In these conditions, therefore, there is considerable danger in childhood, but the statement of the author that "only 13 per cent of severe adult deafness is the result of middle ear disease beginning in childhood" is probably correct. Nothing I have said, however, should be construed as a lack of appreciation on my part of the important work that has resulted from the periodic examination of school children.

I am much gratified by the emphasis laid on the frequent indiscriminate and useless treatment in cases of otosclerosis or, more properly, otospongiosis. It is my opinion that when a diagnosis of otosclerosis is made, the proper thing to do is to give the patient good advice, so that he will not waste time, money and hopes visiting one otologist after the other, having one manipulation after the other, which may make the condition even worse, or having operative work done to help the impairment of hearing. If there are mechanical or infective processes in the nose or throat and these conditions are causing trouble in and of themselves, the pathologic condition should be treated, but not with the expectation or promise that they will have any effect on the otosclerotic processes.

In the experience of the author there have been some cases in which there has been overtreatment or improper treatment; these cases emphasize this point. I believe it most timely and important to have the essayists call attention to these various aspects in the handling of cases of otosclerosis, which facts many otologists, including myself, have urged for a long time.

DR. WENDELL C. PHILLIPS, New York: I wish to call attention to the question of nomenclature. There has to be some sort of a nomenclature in every phase of medicine and disease. So far as my experience goes, the best basic division of these patients is, first, the deaf and, second, the hard of hearing or those with impaired hearing. The deaf should be described as those who lost their hearing before the acquisition of speech, or those who were born deaf, the congenital deaf. That group is very small. Those are the persons we used to speak of as deaf and dumb, but today we describe as the deaf. They themselves have purposely desired that that should be the classification.

There are only 1,000 children of 120,000,000 inhabitants of this country today in schools for the deaf. Many of those children have sufficient hearing to permit their being in schools for the hearing, with the necessary instruction in lip reading, but they are put there through politics.

According to the White House Conference, the estimate was that the so-called deaf in this country number not more than about 40,000 to 50,000 inhabitants, and the rest should be called the hard of hearing. I greatly regret that in this paper the proper nomenclature has been neglected. I know that Dr. Shambaugh will be only too happy to arrange his paper so that he will not be calling the hard of hearing the deaf, because there must be some sort of a classification. It ought to be so in a paper as scientifically arranged as this one.

In examining the make-up of the leagues for the hard of hearing, we should expect them to include a large proportion of otosclerotic patients, because that would be where we should look for this condition.

I have never been able to fall in with the idea of Dr. Shambaugh and some others that the proportion of genuine otosclerosis is nearly as large as the proportion that is given by others who lean toward the otosclerotic theory so strongly.

I am very happy that one of the discussers called attention to the necessity for pathologic examination of the temporal bones of these people. I think that it is most regrettable that Dr. Fowler is not here this morning, because probably no one is getting so many temporal bones of cadavers as he is. I do not wish to quote Dr. Fowler, but I know that he considers that the proportion of genuine otosclerotic patients is comparatively small. In this he does not agree with men like Dr. Shambaugh, who place it very high. It is a question that must be settled, just as the discusser said, by an examination of the temporal bones.

DR. THOMAS J. HARRIS, New York: The subject of nonsuppurative disease of the ear in its relation to deafness is perhaps the biggest subject before the otologic profession today. It centers around what has been discussed this morning, the subject of otosclerosis. I think that we have reached the point where we are not going to be able by mere clinical procedures to determine the real cause or causes of the particular case of impaired hearing, assuming and ruling out and leaving out the question of the congenitally deaf.

The work of Dr. Fowler has been mentioned. I should like to mention the magnificent work that Dr. Crowe has been doing at Johns Hopkins Hospital; it concerns hundreds of temporal bones that he has cut. It leaves one with the conclusion (it does me anyway) that we have a great deal to learn clinically before we can make any diagnosis positively, at least except in occasional cases, of what is true otosclerosis and what is not, other than by examination with the microscope.

The first thing I should like to refer to is the close association between disease of the middle ear and otosclerosis. It is as yet unproved whether the disease of the middle ear is the cause of the otosclerosis or, as we have for many years thought, a mixed infection, a joint infection, with only coincidental relationship.

The work of Dr. Francis Emerson of Boston would give the indication that it is fairly probable, as the author has said, that there is a comparatively small amount of progressive deafness in otosclerosis. Dr. Emerson has shown that in all cases we are dealing with perception deafness due to infection that spreads, in most instances, as he believes, from the throat, the middle ear being involved only late in the infection.

The author has failed to mention a point that I think should receive a great deal of emphasis, namely, the audiograms. In the work of Dr. Crowe and Dr. Fowler, all of their cases have been studied with the audiograms, with considerable findings; the diagnosis is not made from the audiograms, but the observations help very much in making the diagnosis.

Heredity enters into the question only part way. There is another factor, and that is the last point that I wish to make. I was very much impressed with the paper of Dr. Fowler in which he showed, in his controls, that in the cases in which there were normal ears, the accessory sinuses of the nose were comparatively normal, but in a very high percentage of the cases of progressive deafness, the cases of the hard of hearing, so far as the audiograms could be interpreted, there was or had been previous disease of the accessory sinuses of the nose. This point, I think, is a fresh point and one that we should bear in mind.

I am sure, speaking for Dr. Fowler, Dr. Shambaugh and Dr. Crowe, that by the united and progressive work of us all, particularly in having sections made of the temporal bones, a solution will be reached as to just what is the cause or causes of progressive deafness.

DR. GEORGE E. SHAMBAUGH, JR., Chicago: Bezold had more chance to confirm his diagnosis pathologically than we have had, because of the frequency of autopsies. He diagnosed otosclerosis more than a thousand times in his private practice. He found a family history of otosclerosis in only 58 per cent of his cases. We were able to find such a history in 78 per cent of our cases.

AGRANULOCYTOSIS: APPEARANCE OF THE EARLY PHARYNGEAL LESION. DR. JAMES B. COSTEN, St. Louis.

The recognition of this rapidly fatal disease may be delayed because its prodromal symptoms—headache, malaise, sore throat and fever—are common to so many ordinary attacks. The total course, with recovery or death, occurs within a period of two weeks. The data of Taussig, reviewing 314 cases, are quoted, to show a large decrease in mortality among patients treated with high voltage roentgen therapy in the early, hopeful stages.

Numerous authors are quoted, showing the frequent description of a lesion on the anterior pillar; some of the lesions were incised because of abscess or cavity formation. In 2 of the 3 cases presented here a lesion was present, and it was

noted that it represented merely a solution of tissue, a part of the necrotic process which was proceeding on the surface of the tonsil. A probe may be passed through this spot to the necrotic area. A similar undermining of tissue away from the mucosal margin occurred on the gums in these cases. The sign is regarded as constant in the mucosal lesion of agranulocytosis, and may be of value in prompt recognition of the disease.

All three patients gave a history of the regular ingestion of large amounts of drugs for years. The patient who recovered had a definite relapse after her first discharge from the hospital and the resumption of large doses of a sedative drug. These cases are rather good evidence of the depressing effect of drugs on the function of the bone marrow.

DISCUSSION

DR. JOHN J. SHEA, Memphis, Tenn.: I enjoyed Dr. Costen's presentation, for I believe that there is a form of agranulocytosis which has a definite lesion in the throat. A toxin may stimulate or paralyze the hematopoietic system. The action of the roentgen rays is like that of these toxins; a small dose stimulates, while a more massive dose arrests the activity of the blood-forming tissues. I have seen cases of agranulocytosis produced by infections other than of the throat, by arsenic and as the result of multiple fractures of the long bones. These cases are to be distinguished from the agranulocytic angina described by Turck in 1907 and by Schultz in 1922.

On May 6, 1929, I saw in consultation a woman, aged 51, an American, at which time she complained of weakness, sore throat and fever. One sister had died of a blood dyscrasia (myelogenous leukemia). One uncle had died of carcinoma of the throat, and one of her grandmothers had died of a malignant condition of the eye. The patient had frequent attacks of tonsillitis during childhood; the right tonsil had been incised at the age of 21 years, with good after-results. During the past summer, she had malaria but had not taken quinine. Eight months before examination she had neuritis and pyorrhea, and her remaining teeth were extracted.

Three weeks before examination she had fever with no localizing symptoms. Quinine was administered, but was discontinued because of urticaria. One week later a severe sore throat developed, and the family physician gave her diphtheria antitoxin, as both tonsils were covered with a pseudomembrane. For one week her throat was sore, and the temperature rose each evening to as high as 104 F. A few days previous to admission she had diarrhea and pain in her knees.

The general appearance was that of an acutely sick woman with an icteroid tinge. There was herpes on the nose and lips. There were no teeth, and the alveolar processes were normal. The tongue was heavily coated with a white fur. The pharynx was infected. There was a necrotic ulcer on the anterior pillar of the left tonsil, and the posterior pillar of the same tonsil was displaced by a swelling from behind, which was probably a gland. The tonsils were clean, but had a cadaveric appearance. The anterior glands of the neck were palpable and tender.

There were numerous bronchial râles. The heart was normal. There was a generalized tenderness of the abdomen, but the liver and spleen were not palpable. The genitalia showed marked irritation, with an erosion on the left vulva. The knees were tender but not enlarged, with normal mobility. There was a small erosion of the rectum.

Examination of the blood showed: red cells, 3,240,000; white cells, 1,200; polymorphonuclears, 10 per cent; large lymphocytes, 10 per cent, and small lymphocytes, 80 per cent.

Urinalysis gave negative results.

Smears from the lesion of the throat were rich with the bacteria of Vincent's angina. A culture made from the same region was negative for diphtheria bacilli.

The treatment consisted of three transfusions of the whole blood, averaging 200 cc. High voltage roentgen therapy was applied to the tonsils, the glands of

the neck, the pelvic girdle and the long bones. A blood count was made daily, and descended as low as 500 leukocytes, with an average of 10 per cent of granular cells.

The lesions of the mouth became more lymphocytic, and similar lesions appeared in the rectum. Severe diarrhea ensued from similar intestinal lesions, and the patient gradually grew weaker and died.

Autopsy revealed that the bone marrow was aplastic and consisted mostly of fat, with a few small areas of marrow cells, consisting chiefly of lymphocytes and other mononuclear cells. Microscopically, the tonsils showed atrophy of the lymphoid structure.

DR. PINKNEY V. MIKELL, Columbia, S. C.: My chief reason in coming to Chicago was the appearance of this subject on the program. My case has certain features in common with Dr. Costen's, and its course might have changed if the factors which were so evident late had been recognized early.

Mrs. A. S. M., aged 55, of Scotch-Irish extraction, was admitted to the hospital on June 15, 1931, with an ulcer on the left labia. Her past history was irrelevant. She was the mother of four children, who were living and well.

In the surgical service, the ulcer was excised; section was made by Dr. Plowden, who reported "a surface ulcer, with base of inflammatory tissue; no suggestion of malignancy." The lesion healed, but slowly, and by granulation.

On June 15, examination of the blood showed: white cells, 6,400; polymorphonuclears, 41 per cent, and lymphocytes, 58 per cent. The Kahn test was negative.

The second admission to the hospital was fifteen months later. The patient had enlargement of the cervical gland on the right side and gingivitis from which Vincent's organisms were recovered. Two loose teeth were extracted, and 0.7 Gm. of neoarsphenamine was given, with much improvement.

On Sept. 14, 1932, examination of the blood showed: white cells, 6,000; polymorphonuclears, 56 per cent, and lymphocytes, 43 per cent.

Eight days after admission the swelling involving the whole right side of the neck was incised; pus was recovered. This wound continued to drain and never healed, in spite of a secondary effort to locate the course of the pus.

On November 6, the patient was again admitted to the hospital, with a temperature of 102 F. Examination of the blood showed: white cells, 1,900; polymorphonuclears, 0, and lymphocytes, 100.

She promptly improved after five intravenous doses of pentnucleotide, and the treatment was continued, the white count increasing to 5,200. She was fairly well for three weeks, but on December 13, a small ischiorectal abscess appeared. The blood picture again failed, and did not respond to any measure. The patient died of bronchopneumonia on December 20.

The connection which my case has is clear. Even though we did not note the failure of the leukocytic response to infection on the first two admissions, we might have recognized the vaginal ulcer and gingivitis. If the typical undermining sign had been familiar, the suggestion of agranulocytosis might have been made early.

Further, in reviewing my case, I believe that the ischiorectal abscess was merely a late stage of tissue necrosis, the origin of which was the vaginal ulcer.

DR. WILLIAM E. GROVE, Milwaukee: I think the hematologists are agreed that the etiology of these conditions is not the same, and that all of the cases that we now call agranulocytosis are not clinical entities.

I had the good fortune, or the bad fortune, to follow up one case through recurrences over a period of two and one-half years. With regard to the initial lesions, I wish to say that they are not always the same, even in the same case.

During two of the recurrences of this condition, the lesions described by the essayist were found. During the third recurrence, the appearance of the mouth and pharynx was simply what one sees in acute pharyngitis of streptococcic origin, with no ulcerations. During the fourth and final recurrence, there were no pharyngeal or oral lesions at all, but there were lesions in the vagina.

I recently saw another case in which the mouth and throat were simply a little red, and no ulcerations appeared.

DR. THOMAS C. GALLOWAY, Evanston, Ill.: Dr. Costen's paper was thorough and accurate, and he did well to emphasize the importance of seeing the early lesion, but I think it must be recognized, after the work of Kracke and others, that when there is a lesion it occurs relatively late in the course of the disease. The essential factors are the exhaustion, probably, of the function of the bone marrow and the recognition of the so-called neutropenias short of ulcerations; necrosis should be determined by repeated blood cultures in persons with low grade sore throats who have unexplained fatigue and exhaustion.

I am sorry that Dr. Costen did not emphasize the importance of chemical therapy, as I have seen two patients who made no response to transfusions and roentgen therapy and, since that time, four patients, one of whom died, however, of hemorrhage, in whom there was a most dramatic response to the administration of adenine sulphate and pentnucleotide. I think that every one who is likely to see these cases should bear in mind the importance of these drugs.

HEMORRHAGE FROM PHARYNGEAL AND PERITONSILLAR ABSCESS: REPORT OF CASES; RÉSUMÉ OF THE LITERATURE, AND DISCUSSION OF LIGATION OF THE CAROTID ARTERY. DR. SAMUEL SALINGER and DR. S. J. PEARLMAN, Chicago.

This article was published in full in the October, 1933, issue of the ARCHIVES, p. 464.

DISCUSSION

DR. SAMUEL IGLAUER, Cincinnati: Hemorrhage secondary to infection in the pharynx constitutes one of the most serious and urgent conditions encountered by the laryngologist. The necessity for a correct diagnosis as to the source of the bleeding and for prompt intervention to control the same has been thoroughly discussed by the essayists in their very complete paper. As they have indicated, spontaneous hemorrhage or bleeding after an abscess has been incised and drained is more frequent than is commonly believed.

In my own experience I can recall approximately ten cases of this character, some of which resulted fatally; in others recovery took place either with or without ligation. The bleeding may occur in three ways: (1) as a fulminating single hemorrhage resulting in immediate death, (2) in the form of recurring hemorrhages or (3) in the formation of a false aneurysm as manifested by the formation of a pulsating hematoma in the pharynx.

Clinically, it is often impossible to determine which vessel is eroded, but the statistics of the essayists prove that in the vast majority of cases the bleeding arises from the internal carotid artery. The patient is more fortunate if a false aneurysm develops, because this might indicate a smaller opening in the artery and permit a more favorable interval for surgical intervention.

While it is true that some of these patients may recover without ligation of the vessels, the chances for recovery without operation, as pointed out by the essayists, are only 23 per cent, while with ligation 65 per cent recover.

When it has been determined that ligation is necessary, the next question arises as to which vessel should be tied. As already stated, the severe hemorrhages usually arise from the internal carotid artery and, less frequently, from the external carotid artery or its branches. If time and the condition of the patient permit, the plan proposed by Skoog may be followed. He advises exposure of the common carotid artery near the bifurcation and exploration to visualize, if possible, the vessel from which the bleeding comes. This, of course, is often very difficult owing to the infiltration of the neck, and if this procedure is not feasible, then the common carotid artery should also be ligated, because if the internal carotid artery is leaking, the collateral circulation from the external carotid artery may lead to secondary hemorrhage.

I recall such a case which I saw in consultation. The patient, a physician's child, had had a ligation of the common carotid artery, but the hemorrhage recurred and the child finally bled to death despite the previous ligation.

The suggestion of placing a provisional ligature around the carotid artery with an attendant ever present to tie the vessels in case a hemorrhage recurs is an excellent one, but it is important that the attendant never leave the bedside. In a case at the Cincinnati General Hospital a provisional suture was placed by the surgeon, and a nurse was told to pull the ligatures in case of any bleeding and not to leave the bedside under any condition. Early in the morning, at the request of the patient, the nurse went to get him a drink of water; when she returned the patient had died of a hemorrhage. Postmortem study in this case showed erosion of the internal carotid artery.

Surgeons hesitate to tie the common or internal carotid artery because of the possibility of ensuing hemiplegia, and this actually does occur in about 25 per cent of all ligations. There is, therefore, a distinct advantage in exposing the vessels under local anesthesia. In a recent case I exposed the bifurcation, placed a temporary ligature around the common carotid artery and asked the patient to move her arm and leg on the opposite side of the body to see whether there was any effect on the brain. After continuing this for about fifteen minutes, I finally tied the vessel and also the external carotid artery. The patient made an uninterrupted recovery. Of course, one cannot stand at the operating table for an indefinite period in making this experiment.

In conclusion and in explanation of the occurrence of hemiplegia, I should like to refer to the work of Kramer. He found on animal experimentation that, after injecting dyes into the carotid artery, the brain was stained only on the side of the injection, indicating that the circle of Willis does not function for both sides of the brain under ordinary conditions. Therefore, in those cases in which no hemiplegia results, the circle, as well as other collateral vessels, must dilate to relieve the anemia of the brain on the ligated side, and this collateral circulation is more apt to be established in younger persons with more elastic arteries. I also think that transfusion immediately after ligation, tending to raise the blood volume and the blood pressure, helps to obviate the recurrence of hemiplegia.

Dr. O. Jason Dixon, Kansas City, Mo.: In some experiments on the external jugular vein of the dog with viable muscle implants, I thought that I hit on a solution of the prevention of the 25 per cent of complications, namely, the hemiplegia. I found that when viable muscle implants are used in the vein of the dog, as the external jugular vein is larger than the internal, that vein always recanalizes; recanalization of the vein begins to take place about the sixth day, and within three weeks the vein is again carrying its normal amount of blood.

I wrote to Dr. Matas of New Orleans about this, and I also wrote to Dr. Barney Brooks. I was enthusiastic over the observation, and I started to work on the dog, using implants in the common carotid artery. I have been very much disappointed. Up to date I have not been able to prove conclusively that this artery becomes recanalized. I believe it is because I have been putting in the muscle too tightly to control the hemorrhage, as it is an entirely different problem when you begin to work with arteries and veins.

I do think that further experiments will show that viable muscle will be a very good substitute. I have just one other suggestion to make, that is, to tie off the internal jugular vein at the same time you tie off the artery, to prevent anemia of the brain.

Dr. Thomas C. Galloway, Evanston, Ill.: May I show this slide? The interesting thing about this girl is that she lived eight days after her first sudden and profuse hemorrhage of about 400 cc. Unfortunately, I had not had the advantage of having the matter brought home to me as is done in this paper.

I think that this case illustrates that any sudden and profuse hemorrhage of that kind is an urgent indication for immediate ligation of the carotid artery. The artery was eroded high up, just before its entrance into the carotid canal. The

girl had four hemorrhages, averaging 400 cc. and 300 cc., and she died of a hemorrhage of about 500 cc. while the carotid artery was being ligated.

DR. JOHN W. CARMACK, Indianapolis: As I had occasion to review the anomalies of and accidents to the internal carotid artery and the large vessels of the neck a few years ago, this presentation is extremely interesting to me.

Erosions of the large vessels around the pharynx from abscesses are usually due to neglect of drainage of the abscesses. We know that the pharyngomaxillary abscesses remain in that space for a considerable length of time before any great amount of swelling is present externally. In order to drain these abscesses at the proper time, we must drain them long before fluctuation occurs in the neck, because the pus is inside the fascial planes of the neck and does not produce fluctuation.

We can eliminate many of these erosions if we recognize pharyngomaxillary abscesses and peripharyngeal abscesses early and drain them before erosion occurs.

DR. SAMUEL SALINGER, Chicago: The point that Dr. Iglauer made about ligating the external carotid artery when the common carotid artery is ligated is well taken. I mentioned it in the paper for the reason he stated. When the common carotid artery is ligated, the back pressure from the external carotid artery may pass the bifurcation, and hemorrhage occurs through the erosion of the internal carotid artery.

Why do we not ligate the internal carotid artery? Because in most cases, on account of the pathologic changes in the neck, it is difficult to reach a healthy portion above the bifurcation. It would be advisable if it could be done.

I also went into detail in the paper regarding the modus operandi of hemiplegia—how it occurs, why it occurs, the different types and the difference in the prognosis.

Dr. Dixon mentioned the muscle implant. I refer to a number of experiments that have been made over a period of years seeking natural methods of obliterating the vessel without damaging it.

Also, with reference to ligating the internal jugular vein at the same time, this procedure is recommended by some, and others say not to ligate the internal jugular vein. There certainly is no definite evidence one way or the other.

A MOVING PICTURE OF THE EXTERNAL ETHMOSPHENOFRONTAL OPERATION. DR. W. LIKELY SIMPSON, Memphis, Tenn.

A motion picture demonstration of the operative technic was presented.

DISCUSSION

DR. E. C. SEWALL, San Francisco: This has been an excellent demonstration. I congratulate Dr. Simpson.

I differ in certain respects. In regard to removing the oblique muscle, this does not seem to be necessary. If the periosteum is taken off, the muscle is taken off with the periosteum. I differ also in regard to acute cases. I do not think that osteomyelitis occurs except in cases in which partial operations are performed. Therefore, I consider this the operation to perform in all acute cases, because it gives clean, thorough drainage.

In regard to treatment of the sphenoid sinuses, I believe that it is possible, and really better, to take off the floor of both sinuses, because in that way the drainage is wide enough so that granulation tissue does not spread across and interfere with the cure. I do not use packing after operating. I use skin clips, which I think are preferable to anything else because there is not the sucking up of infection from below.

Thirty years is a long time, but how short it has seemed in measuring the progress of surgical treatment of the accessory sinuses! During that period there has been recorded a succession of remedial endeavors, beginning with topical applications, running the gamut of radical surgical effort and represented today by opinion divided as to the progress that has been made.

On adapting surgical principles in the treatment of purulent foci, it was natural to follow the discovery of pus by an attempt to institute so-called drainage. Topical applications gave disappointing results, but the surgical era has not brought the desired panacea. This must be granted by the most sanguine.

As I was born with technical inclinations toward surgery, improved by study under Killian, the master, I early joined in the dream of mastery of the sinus problem by the application of surgical principles. While it is not fair to admit failure, all will agree that these early dreams, which persist in recurring, are troubled and occasionally may be likened to a nightmare. How can we evaluate what has been done? Surely endeavor has taught us much.

I have grown to recognize the almost universal prevalence of sinus infection, to respect its vicious character in certain cases and to fear its complications. I do not hold with those who minimize the effect of that infection on the general economy. In some quarters the terms "focus of infection" is unfashionable. While not as well qualified as many to speak on this subject, I cannot but record my opinion that the principle, long recognized before the catch phrase was coined, brought to medicine one of its greatest truths.

Certainly the causal relationship between sinus infection and other disease cannot be discredited because no evidence of sinus infection has been found in a cursory examination. Still less can the association be disproved because the patient has been operated on. I have seldom seen reports in which statistics were based on convincing evidence of lack of sinus infection—granting the questionable assumption that all disease of the sinuses can be determined by present methods—or in which the complicating disease of the sinuses was entirely eradicated—again conceding the neat conclusion that it can be completely cured.

Autopsy has brought to light unsuspected disease, particularly in the sphenoid and ethmoid sinuses. This will be seen more conclusively when the sinuses are regularly examined grossly, bacteriologically and histologically.

The better understanding of general, allergic and dietetic therapy finds a sincere appreciation at the hands of the rhinologist. No one is more anxious than he to avoid the mutilating methods that have been developed in the past few decades. It is my privilege to work with a service that furnishes a point of view that I can harmonize with the pathologic process as I see it. I cannot separate the etiology of sinusitis from lack of drainage. I believe that sinusitis, as we understand its pathologic process, is always more than allergy. If allergy has been a predisposing cause, it is through interference with drainage. Relief from the allergic swelling may in turn so benefit the drainage that the sinusitis heals. I believe that the presence in the discharge from the sinus of sufficient numbers of leukocytes distinguishes sinusitis complicating the allergic state. This may be difficult to estimate when the leukocytes are few, easier when they are numerous and certain when polymorphonuclear leukocytes are abundant. More power to the nonsurgical endeavors! At present they stand more in need of our prayers than surgical methods.

While conservatism is in fashion and is generally sanctioned in view of the complexity of the entire problem, nevertheless, there are certain conditions necessitating operative intervention. Prominent among them are incapacitating pain, acute inflammatory manifestations and complicating disease of the eye or the brain or general disease, demanding removal of the causative pathologic process. To care for such conditions, an operative procedure with a long ancestry has been developed. This Dr. Simpson has ably described. For this excellent operation we must be grateful to the era of radical surgical endeavor.

The question whether all diseased sinuses can be cured by operation is important. There is lack of agreement on this point. Many believe that in many instances it is impossible to eradicate the disease through surgical methods, particularly drainage. I have often debated this question with myself, and frequent discouragement has produced an impression that has only gradually been removed. I have now come to believe that the question resolves itself into what is meant by

surgical drainage. If one means making a hole in a sinus in the hope that all the disease will find that hole and escape thereby, one has a common example of the so-called drainage our methods frequently provide. If, on the other hand, one means the removal of the entire floor of a sinus so that there is no place for accumulation, then one has true surgical drainage in its logical sense.

I have reason to believe that such drainage, which is only occasionally furnished by the most radical methods, will cure sinusitis. One must limit the meaning of cure to freedom from discharge. There is sufficient evidence in the literature to support this belief, just as the cures that we all obtain in some cases bear it out.

Special emphasis has been laid on the inadvisability of removing the sinus mucosa. It has been written that removal of the sphenoid lining leads to a granulating mass that exaggerates the original pathologic process. I have been forced by complicating disease to reoperate on such a sphenoid sinus until the whole discouragingly thick floor has been removed to have it heal as clean as your hand. What else could it do without a floor? I have seen the same discouraging picture in an antrum which has been repeatedly operated on with so-called radicality. I have found such an antrum full of adhesive, pocket-forming, fibrous tissue and have seen it get well under my eye because the dependent opening at the alveolar process was kept widely open, by a rubber tube as large as my thumb.

This is no plea for surgical methods. It is simply a statement that if these methods are permitted by the anatomic conditions to furnish dependent drainage, the cavity can no longer harbor secretion.

DR. FRANCIS E. LEJEUNE, New Orleans: This excellent presentation conveys in a most thorough manner the details of the ethmofrontal operation as advocated by Dr. Simpson.

The majority of my operations are performed with the patient under general anesthesia, and I must frankly confess that I cannot obtain the desired result with an incision as small as that advocated. I believe that the incision is totally dependent on the size of the frontal sinus. Under general anesthesia, hemorrhage from the incision is very free but easily controlled by making a superficial incision first through the skin only and injecting a 0.5 per cent solution of procaine hydrochloride with epinephrine into the deeper structures. The incision is then carried to the periosteum, and very little trouble is experienced with bleeding.

I thoroughly agree with Dr. Simpson that all the mucous membrane should be removed from the frontal sinus, and I believe that obliteration of the external angle is to be desired and minimizes possible recurrences. The late Dr. Lynch, in his classic description of his technic of operation on the frontal sinus, laid great stress on this point and stated that recurrences were usually found to be due to the harboring of secretion and infected tissue in the external angle.

The patients with ethmofrontal sinus injection who come under my observation either have had or are subjected to various intranasal procedures in an attempt to clear up the existing pathologic process. An external approach is resorted to only when intranasal surgical intervention fails to accomplish the desired results, and in practically all cases, the middle turbinate has been removed at the time of the intranasal procedures. In a subtropical climate, such as we in the far south live in, no discomfort is experienced from this procedure, and opening of the sphenoid sinus is facilitated.

I must take issue with Dr. Simpson as to the final closing of the wound. I believe it to be a cardinal principle of surgery that all incised tissue should be replaced in its original position, and for this reason, I insist on suturing the periosteum and believe that this helps to maintain the normal position of the orbit and its contents.

I feel as Dr. Sewall does about the superior oblique muscle. There is no necessity for cutting it. Elevation of the periosteum automatically detaches the attachment of the superior oblique muscle.

My experience with curetting of the frontal sinus has so far been rather happy. I have had occasion to watch a large number of chronic cases in which curets were used with a fair amount of vigorousness. In no cases have I seen resulting osteomyelitis.

DR. JOHN B. POTTS, Omaha: There is only one thing I wish to state, and that is that in the microscopic examination of the pathologic membranes that have been removed in our work at the University of Nebraska, we found that the chronic hyperplastic type membranes are permeated, entirely through the membrane to the base as far as the bone, by bacteria. This, I think, is the point that clinches the argument that it is necessary to remove the membranes, and is the real essence of the question as to why intranasal drainage, with only an opening for drainage, fails. There is no more sense in thinking that a membrane of this type will heal, become a normal membrane, than there is to think that a tonsil with the top of it cut off will heal and become a normal tonsil. The chronic infected tissue must be removed in order to eradicate the disease entirely. If that is done, as Dr. Sewall says, nature will furnish a new membrane, healthy, smooth, clean and uninfected, and a symptomatic cure is obtained.

DR. W. LIKELY SIMPSON, Memphis, Tenn.: In answer to Dr. Sewall, I feel very strongly that I should not operate in a case of acute fulminating sinusitis. I do only drainage in those cases. When a frontal or a sphenoid sinus is full of pus, I feel much safer in draining it by external drainage. If the frontal sinus is inspected, I let it go on, two or three months, and then clean up the infection.

As for skin clips, I do not think it matters how the external wound is closed. I use the dermal clip; I take it out in twenty-four hours, and there is no effect on the skin.

In answer to Dr. LeJeune's discussion, I seldom if ever put artery forceps on any part of the wound in performing an operation on the frontal sinus. It is not once in ten cases that I ever tie a vessel. The vessels bleed very little. I pack them off a little if there is any bleeding, except, I should say, the anterior ethmoid vessel; I mean that I do not tie the ordinary vessels in the soft tissue. There is practically no hemorrhage during the operation.

I invariably leave the turbinate bone, unless it has been removed at a previous operation.

In regard to replacing all the subcutaneous tissues as they were, I formerly drew up the superior oblique muscle with one catgut suture, or probably several subcutaneous catgut sutures, but a few infections occurred, and I no longer use any subcutaneous sutures whatever.

I probably did not make it clear about the superior oblique muscle. I elevate it just as the discussers do, but I have found that I can elevate it with a knife turned upward, and it is elevated with the mucous membrane. I do not mean that I cut the muscle; I cut the attachment of the muscle away from the bone. I do it with a sharp elevator or usually with a knife, turning toward the muscle.

I try to leave the nose as nearly as it was before the operation as is possible. I do not remove any turbinate tissue. After the operation intranasally the appearance of the nose is the same as it was before operation, as far as the turbinates and so on are concerned, and externally there is no deformity worth mentioning.

THE PERSISTENCE OF APPARENT SINUS PAIN AFTER MULTIPLE OPERATIONS. DR. SAMUEL R. SKILLERN, Philadelphia.

This article is based on the opinions of sixteen prominent rhinologists from all sections of the country, thus eliminating local teachings and climate as a factor. The opinions of these rhinologists regarding the etiologic factors of persistent postoperative sinus pains may be divided as follows:

1. Incomplete operation (seven).
2. Disease elsewhere than in the sinus attacked (six).
3. Systemic disease giving symptoms of involvement of the sinus (five).

4. Synechiae or scar tissue blocking drainage or enmeshing the nerve, with painful contractures (five).

5. Direct injury to, or interference with, a nerve (four).

6. Operations on the ethmoid or turbinate for insufficient reason (four).

7. The patient's type of nervous reaction to pain (three).

8. Neuritis of the fifth cranial nerve (three).

9. Atypical neuralgia of unknown origin (three).

10. Reaction to insult of the periosteum and bone, especially about the foramen (three).

11. Injury to the sympathetic plexus (two).

12. Diversion of normal air currents within the nose (one).

To these opinions I should like to add lighting up of an unsuspected focus in an obscure cell, silent abscess in the frontal lobe, localized meningitis, spreading of infection into traumatized tissue and infection of the newly regenerated mucous membrane.

DISCUSSION

DR. BURT R. SHURLY, Detroit: The problem of pain after a series of operations is probably one of the very important problems that belongs to the otolaryngologists, because if pain did not bring patients to them, they would certainly have a far smaller number than they have at the present time in this sad economic state of affairs.

The whole question resolves itself into a scientific classification of what sort and variety of pain the patient has, the personal equation of the patient being carefully analyzed and carefully sought.

When a series of operations for disease of the sinus are performed on patients, many of them become, as you well know, more and more neurasthenic, more neurotic and more susceptible to their own internal, intrinsic reactions that we call symptoms.

When in that state the patients become very susceptible to their own introspection. I believe that when we see such a patient there is nothing more important than determining whether there is some outside, systemic or remote etiologic factor which must be carefully thrown out time and time again, because many of these patients, under the present psychology, order an antrum wash just as they would order baking soda in the grocery store or acetylsalicylic acid in the drug store. They have been educated, under the present modern psychology, to order an antrum wash, a hypodermic or some particular kind of treatment. In other words, a certain number of these patients with persistent pain belong to a particular type that we call tickers. They have a habit spasm which is steadily and rapidly developed. Some of them have that hang-over and hand-over of the early days of tickers when they went along to school and hit one post after the other or one telegraph pole after the other, all the way alnog, and they were not happy or satisfied until they had accomplished that same feat to and from school. There is something to that problem. There is something to the changing psychology, to the changing conditions of patients, which emphasizes pain and puts the postoperative treatment in the place where the operation was before. I believe that we have to understand that situation and orient it, throw it out and classify it; so that when we discuss the problem of pain we encounter those that deal with the imaginary, the so-called neurasthenic, type and the genuine, absolutely unquestionable pain that many patients have.

The surprising report just read from Frazier's clinic of 148 cases of atypical neuralgia, in which they were unable by a whole series of treatments of various sorts, including operative intervention, alcoholic injections and, in fact, all of the latest methods, to do anything to relieve the pain of the patients deals with a special type of case.

There is, of course, the final analysis which belongs directly to our specialty, and that is a careful, common sense, good judgment study of the individual case

that is shown by roentgen and every other possible examination to be the result of an incomplete operative procedure; that patient has not had the full value of surgical technic; there is a problem of pus which has not been eliminated, and incomplete drainage that has not been taken care of. Of course, these patients must be given the benefit of complete scientific care and operative procedures.

Dr. Frederick E. Hasty, Nashville, Tenn.: Early in my experience I had three years' training in a psychopathic hospital. I was early impressed with the frequency with which patients coming to that institution, after they had reached a well developed state of psychosis, would give as their early history pain about the head. This same idea will hold with the development of neuroses, and we cannot well separate neuroses and psychoses. They must all have a beginning and must go through the different stages of development.

A smart thing to do is to allow the patients to come before you, put them at ease and say: "Now, tell me your complete story in your own way, as you see it." Then apply that history to what should be a typical history of a sinus condition. The patients will frequently classify their own conditions, or will at least give an indication of what one should beware. If their conditions belong in the class of psychoneuroses, slip them over to your friendly enemy. He will operate on them, but he will do his reputation no good.

That is one of the things, in my opinion, that is wrong with us today in the eyes of general practitioners and of the public as a whole, and is what to their minds is partial justification of the saying "once an operation, always a sinus condition." The truth is that the majority of such patients did not primarily and essentially have sinus diseases. There is much that we could learn about neurology that would be of inestimable value to us in rhinology. We should, and we really must, learn to utilize all available assistance from neurologists. If a patient does not have a condition that clearly comes within the classification of a sinus disease, we shall be wise to be cautious.

Dr. Ferris Smith, Grand Rapids, Mich.: In discussing this paper, I am assuming that we are talking about multiple operations on sinuses in cases in which a definite diagnosis and indication for operation have been established.

There is no longer excuse for multiple operations in the sense of Dr. Skillern's paper. We have the technical means of completing operations on one or all of the sinuses in a single procedure. It is no longer necessary to attempt to complete in our offices that which should have been accomplished in the operating room.

A small proportion of these patients are operated on because of a mistaken or incomplete diagnosis. They should not be operated on at all. The large majority of these persistent sinus pains result from retained pathologic changes. Of the fifteen specialists answering Dr. Skillern's questionnaire, nine are of this opinion, and three believe that the pain results from nerves incarcerated in the scar. This cannot be of frequent occurrence, or, otherwise, this sequel of incision would prevent many of the surgical measures performed on all parts of the body.

It is my opinion that these pains depend on inflammatory processes in the linings of the sinus or the bony walls, which result in localized pachymeningitis or venous congestion, such as produces the retrobulbar pain of Gradenigo's syndrome. The disappearance of this pain and other symptoms occurs with the removal of the pathologic changes in the mastoid which occasioned the venous congestion about the petrous tip. This is equally true in the sinuses.

Dr. Skillern is discussing chronic pathologic changes. Such changes in this locality are only occasionally completely removed by the practices which are generally in favor. These persistent pains result from the failure completely to clean out all of the involved cavities and to remove their diseased linings. It is impossible to do this safely by intranasal methods. It requires the most meticulous care to accomplish this under direct vision in a bloodless field.

In many instances the inflammatory process in these lining membranes enjoys resolution in the presence of free drainage and ventilation; the circulation is restored to normal; the venous congestion disappears; the pain ceases. Unfortu-

nately one cannot judge the chronically infected membrane which will approach normal resolution and cease to produce symptoms. It is foolish to advocate the removal of part of such a membrane with the expectation that the balance will approach the normal. In several hundreds of microscopic examinations, we have never seen any part of such a membrane that was fit for retention.

There are headache and pain of varying degree and duration after every complete operation on the upper group of sinuses. They persist for from one to fifteen days, with an average of five days. If these symptoms precede the operation, they are temporarily aggravated by it.

In operating by the direct method on patients who have had multiple operations without relief of various symptoms and signs, one frequently observes cells which have not and could not be exenterated by any operator following the intranasal route. These are periorbital and retrobulbar cells and slitlike cells along the attachment of the middle turbinal. We recently operated on a patient with such cells in Dr. Skillern's clinic. She had had in excess of twenty operations without relief of her complaint.

There are several abnormal pneumatizations which cannot be dealt with by the methods usually practiced. We have drained the crista galli in six cases, with complete relief of frontal symptoms.

The sphenoid sinus is a very frequent source of these unexplainable pains. This applies not only to the sinus itself, but occasionally to its basilar process. The pains are not typical and may be distributed anywhere in the head or neck. The front wall and floor of the sinus should be removed and the lining completely obliterated. Search for the various pneumatizations which frequently occur in this bone should be made. This cannot be thoroughly accomplished intranasally.

I wish to present the roentgen study of a patient who had eight operations on the sinuses without complete relief of symptoms. Her chief complaint for a year had been low grade pain behind both eyes and in the back of the neck with periods of intense exacerbation. The plate shows marked infection (osteomyelitis) of the basilar process of the sphenoid sinus and the body of the occiput. The floor of the sphenoid sinus was removed and a ¼ inch (0.64 cm.) burr was driven upward and backward through these two bones to the dura over the medulla. It was not necessary to incise the dura. The pain subsided on the fifth day in the presence of free drainage; the venous engorgement had been relieved, and the circulation was approaching normal.

DR. WILLIAM V. MULLIN, Cleveland: Without prolonging the discussion, I might say merely that I should always want to present these cases, not as a sinus problem, but as a diagnostic problem, and that the patient should be examined in all departments of the hospital. Then I should like to submit the report of one case that will help to make my point.

A man, aged 51, had pain in the right shoulder for one year (1931). He had undergone a right nephrectomy eight years previously. A malignant condition of the humerus was suspected. He was treated by roentgen therapy, with marked relief from his pain. At that time the right nostril was found blocked with a large polyp. This was removed, and the right maxillary sinus was irrigated three times; following that treatment the sinus became clear, and the patient was discharged from our department. However, he lived out of town and returned in three months with a very acute suppurative condition—pansinusitis on the right side. He was then put in the hospital and kept under observation for four days. The pain was so localized over the right frontal region and over the right side of the face that the sinus condition was very evident.

The patient had haziness of the optic disk, but no edema was noted. A spinal puncture was done. He was given a complete examination. The spinal fluid was under pressure, and was amber. Meningeal irritation and also an acute sinus infection on top of a malignant condition were suspected. He vomited several times, and had very intense pain.

Operation was decided on, and an external fronto-ethmoid operation was performed. I assure you it seemed extremely necessary. There was a very large

amount of pus which came out under tension. The patient had complete relief from pain for four days. He was a very happy man and told us so. At the end of that time he had a return of the pain in his shoulder and a return of his headaches. On the eighth day he became drowsy. Sensory aphasia developed. In the meantime we had received a pathologic report on the operation on the kidney which we had written for, and the report came back that that kidney was removed because of a hypernephroma.

On the tenth postoperative day the spinal pressure was 650 mm. of water. A ventriculogram was then made, which showed a space-filling lesion on the left side. Because of the very severe pain, a frontal craniotomy was performed, which gave relief for a while. The patient returned home, where he died. Autopsy revealed the tumor of the brain found at operation which was not able to be removed, and the pathologic report of the tumor was a hypernephroma occurring nine years after the original operation on the kidney.

DECOMPRESSION OF THE FACIAL NERVE: PHYSIOLOGY OF THE SEVENTH AND NINTH NERVES AND MOVEMENTS OF THE LIDS IN FACIAL PARALYSIS. DR. EDWARD CECIL SEWALL, San Francisco.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. HARRINGTON B. GRAHAM, San Francisco: Of course, I am very much interested in this subject, as everybody is. I think that these cases should be reported because this line of work is entirely new, and we should know more about it.

Dr. Sewall has given an interesting discussion of the anatomy and physiology of the region. The only thing I wish to add is a description of one case that I saw in the very early history of this work. A patient came to me from the San Joaquin Valley after an attempt at some kind of an operation on the attic through the external auditory canal had been made by a general practitioner. He had total paralysis of the face. I performed a radical operation on the mastoid, and during the process of the operation found that the facial nerve had been thoroughly frayed at the promontory wall. The only thing I could see that could be done, as the patient had perfect hearing, was to take the frayed ends of the nerve and tease them together as best I could into a bundle and lay them up against the promontory wall. I did this with the hope that something would happen. It did.

I told the patient that he probably would not get return of function, but within eight months he came back from the San Joaquin Valley smiling all over, which was a very great joy to me as well as to him. I think that when facial paralyses occur a number of hours after the operation, all that can be done is removal of the bone from the nerve, or decompression. I do not think that we should be too anxious about the case for some time, because the return of function is delayed, regardless of the reports that have come out in recent days of the return of function in two or three months. Eight months is not an unusual period of time to wait for return of function, and during that time massage and electric treatment certainly should be instituted in order to help the return of function.

The last word certainly has not been said concerning surgical work in the repair of the facial nerve, and we are all hoping for more reports, especially as far as function following the operation is concerned.

DR. ELMER W. HAGEN, Chicago: There is one point in anatomy that Dr. Sewall mentioned and I should like to discuss briefly. With regard to the case of tic of the ear, Professor Herrick at the University of Chicago informed me the other day that there is a small branch of the ninth nerve which goes to the tenth nerve, so the auricular branch of the tenth nerve can and does carry this connection to the external auditory canal. Section of the ninth nerve intracranially would thus be proximal to where the branch to the tenth nerve comes off, and hence would cure the tic.

This is offered in explanation of the mechanism in place of the explanation based on the fact that the nerve of Jacobson is the nerve involved.

BILATERAL BRANCHIAL CYST. DR. GORDON BERRY, Worcester, Mass.

This case (in a patient, aged 34) is presented for its scientific interest as an embryonic anomaly. Symmetrically placed scars on each side of the neck at the level of the first tracheal ring gave evidence of two fistulous tracts which were present at birth and which had been treated and closed at the age of 20 years. The left tract, however, continued as a long sacculated cyst which finally ruptured through the superior constrictor muscle and, passing up in the fibrous tissue of the capsule and over the tonsil, drained its creamy pus into the mouth through a small pimple-like projection in the plica supratonsillaris. An initial injection of iodized poppy seed oil 40 per cent through this oral opening permitted an exact delineation of the sac, 4 inches (10.16 cm.) long, in the roentgenogram. Excision was readily accomplished, and convalescence was uneventful. A brief consideration of the five visceral arches and the resulting adult structures is given. This case deals with a bilateral remnant of the second arch.

DISCUSSION

DR. LYMAN RICHARDS, Boston: In the human embryo the visceral arches are best developed in the last half of the third week. Their phylogenetic course is seen to advantage in the fish, in which the arches form the gill structures and the spaces between them the gill clefts. Such arches commonly number six, while in the human being there are but five. The ectodermal and entodermal layers lining these grooves between the arches in the human being normally never come in contact, being separated by mesoblastic tissue which prevents any communication. The five arches, by future growth and modification, contribute to the development of certain definite structures in the head and neck as follows:

Arch I (the mandibular arch) differentiates into an upper short (nasomaxillary) process and a lower, longer process (the so-called Meckel cartilage), forming the basis for the lower jaw and in addition for two of the auditory ossicles, the malleus and incus.

Arch II (the hyoid arch) forms the lesser horn of the hyoid bone, the styloid process, the stylohyoid ligament and a portion, at least, of the stapes.

Arch III (first branchial arch) forms the body and the greater horn of the hyoid bone.

Arches IV and V (second and third branchial arches) fuse to form the thyroid cartilage.

Of the external, potential human grooves or clefts, which are permanently open in the fish, the first remains as the external auditory meatus. The others disappear by the twenty-eighth day, with the exception of an occasional remnant in the lower lateral part of the neck (the precervical sinus of His), which may persist somewhat longer.

The internal grooves or pouches have the following fate: Groove 1 becomes the eustachian tube and the cavity of the middle ear; groove 2 disappears except where it contributes to the epithelium of the supratonsillar fossa, and grooves 3 and 4 take part in the formation of the thyroid and thymus glands.

It can readily be seen that in such an arrangement when an external inpocketing approaches an internal outpocketing, some disturbance in development may from time to time result in an abnormal communication between the two. This is the more likely when one realizes that such a communication is the normal outcome in animals further down the phylogenetic scale. It is such a connection, particularly in respect to the second branchial cleft, that leads to the condition of congenital branchial fistula.

Theoretically, any of the following possibilities may occur: 1. Any one of the grooves may persist as a fistula or a tract, the outer portion being lined with ectoderm and the inner portion with entoderm, the origin and termination being in their normal embryonic locations. 2. Any one of the grooves may persist in its

normal pharyngeal position, but by continuation in the cervical sinus it may come to end anywhere on the neck from the top of the styloid to the sternoclavicular joint. This is due primarily to the fact that during the development of the fetus there is a tendency for the second branchial arch to grow downward and overlie the third and fourth arches and thereby to carry the point of exit of the second branchial cleft to a lower point than would otherwise be the case. Hence the exit of such a fistula originating in the second cleft (the origin of almost all such fistulas) may appear at various points on the neck according to the degree of downgrowth of the second arch.

Since one groove may persist in whole or in part, it is often difficult or impossible to state the entire origin of the fistula, but by the time most fistulas are noticed, this matter is of academic interest only. So long as the fistulous tract remains patent throughout, there will be nothing to call attention to it but a moderate amount of discharge through the small pinpoint opening in the skin. The tract, however, may not be patent throughout its entire length. There may be no apparent external communication, as a result most often of some attempts by cauterization or operation to close the fistula. This may result in an internal discharge into the supratonsillar fossa which may go unrecognized until changes effected by a tonsillectomy first call it to attention. Obliteration of the external opening more commonly gives rise to a gradual dilatation in the medial portion of the tract, which as time goes on eventually gives rise to the condition of a branchiogenic cyst. Such a cyst may also occasionally arise from an independent epithelial inclusion or rest, without ever having been associated with a fistular tract.

Such branchial cysts may best be classified according to the character of their lining epithelium: (1) mucoid cysts, containing mucus, arising primarily from the inner portion of the tract, which is of entodermal origin, and showing a cylindric and columnar type of cell; (2) pseudodermoid cysts (lined with desquamated epithelium) essentially of ectodermal origin and showing stratified squamous epithelium. The character of the fluid in the cyst may offer a further basis for differentiation. One may speak of serous or hemorrhagic cysts, each of which may, by becoming secondarily infected, contain purulent fluid.

Either type of lining epithelium may be found resting on a thick band of lymphoid tissue, below which is connective tissue with smooth and striated muscle, infiltrated with round cells. The lymphoid tissue, however, may be abundant, scant or absent and with or without typical lymphoid follicles.

The treatment of such a cyst resolves itself essentially into surgical removal. Incision or drainage of such a swelling will lead only to secondary infection and the formation of scar tissue and adhesions, rendering secondary removal far more difficult. At best the dissection of such a cyst is tedious and often presents a difficult anatomic and surgical problem. Attempts solely to close the external opening of this fistular tract will result either in driving the discharge in toward the throat or in causing a cystic development when not already present. However, as mentioned by Dr. Berry, the recent use of certain sclerosing fluids has met with success in the attempt to obliterate a fistulous tract, particularly when there is not much cyst formation.

Branchial cysts in general cause no symptoms beyond the disfigurement of the swelling. They vary tremendously in size, from that of a small walnut to a mass reaching down to the umbilicus. They may lie superficially or so deep as to be intimately connected with the internal jugular vein and carotid artery. They must be distinguished from tuberculous glands, lipomas, thyroglossal cysts, lymphangiomas and cervical abscesses. There has been noted some tendency to familial occurrence with inheritance, chiefly through the mother. The cysts serve no useful purpose beyond that reported in the case of a circus performer who entertained the sightseers by passing a knitting needle through the pharyngeal opening out onto the side of his neck.

DR. LOUIS K. GUGGENHEIM, St. Louis: The one thing that was left out that would have made the demonstration perhaps a little more complete was some comment as to the origin of these interesting pouches in the human being.

At the end of the second week the endothelium of the intestine grows outward through the mass of mesenchyme in the head end of the embryo to form the pharyngeal pouches. At this stage of embryonic life there is a neural tube, and the intestine is lined with entoderm. The entoderm begins to grow out into the mesenchyme, and in growing out forms the pharyngeal pouches, and the mesenchyme on either side becomes the branchial arch. Finally, when the pouch extends out as far as the ectoderm, there is an arch there and also a pouch; externally there is a groove lined with ectoderm; the pouch is lined with entoderm, and the two do not meet, as Dr. Richards stated.

The first pharyngeal pouch becomes the eustachian tube and the tympanic cavity, and a posterior projection of it, the antrum of the mastoid, which is present quite early, at the time this pharyngeal pouch is completed.

The second pharyngeal pouch forms the tonsillar fossa, and it is from this pouch that these most interesting cysts have formed.

Dr. Frederick A. Figi, Rochester, Minn.: The first case of Dr. Berry's is of particular interest as it shows the more or less serious complication that may arise from an attempt to obliterate the external portion of the sinuses.

At times the cysts become quite large. Frequently they present a more or less difficult differential diagnostic problem. Commonly the patient comes in with a mass situated high in the cervical region underneath the sternocleidomastoid muscle which has flared up acutely on repeated occasions and then subsided. The acute flare-ups may come on immediately following a sore throat or acute pharyngitis or an acute cold in the head, or the flare-up may develop without any such factor entering.

Many of these cysts are taken to be recurring cervical fragments of undetermined origin. Not infrequently they are supposed to be suppurative glands, in spite of the fact that they have been drained repeatedly, so that tissue might have been secured for microscopic study, which would have rendered the diagnosis definite.

When an acute flare-up occurs, one must, of course, await its subsidence, and then dissect out the sinus tract provided the fluid is drained or ruptures through, or dissect out the cyst that remains provided the rupture does not occur.

Dr. Thomas J. Harris, New York: I wish Dr. Berry might, in concluding his discussion, go a little more into the matter of the surgical procedure as compared with the injection of which he has spoken, or rather the injection as compared with the surgical procedure. All of us, or many of us, have had experience with surgical treatment for these cysts, and there have not always been satisfactory results.

Dr. Buckley of New York has pointed out in a number of operations he has done that failure to carry the dissection upward, behind and underneath the hyoid bone, must be avoided, and by removing this bone or proceeding from there he has been able to get a very high percentage of successes.

But when the essayist refers to injections as compared with the operation, he is developing something that is quite new to me.

Dr. Dunbar Roy, Atlanta, Ga.: I have had one case in which there was no connection with the tonsillar fossa but there was a fistulous opening on the outside. About the time I saw the patient Dr. Gifford of Omaha wrote an article on the destruction of the lacrimal sac by swabbing on the inside with trichloracetic acid. I simply took a very small probe with cotton saturated with trichloracetic acid wrapped very tightly around it and inserted it into the sac and through the tract. The patient was cured by the use of the trichloracetic acid.

Dr. Joseph C. Beck, Chicago: It is not my business to discuss, but I cannot help speaking on this subject because at the present moment I have a young woman in my care who had a bilateral congenital condition of the kind described, which was operated on externally successfully, so that there are two linear scars.

She had her tonsils removed after that, and subsequently a recurrent discharge developed periodically from the external auditory canal. She could tell when the attack was coming on, as she felt the tightness in the movement of the lower jaw; then you could watch and see the formation of the fistula, which was within the meatus and which discharged copiously. The attacks were recurrent. I made an incision under the lobe of the ear following one of the attacks and dissected what I thought to be the end of the tract. However, I was mistaken because I have driven the fistula into the throat. She has not only a discharge in the throat but a copious nasal discharge of the cystic material. I now have to look for the fistula somewhere in the region between the soft palate and the eustachian tube.

DR. GORDON BERRY, Worcester, Mass.: The new solution that has been recommended by Cutler and Zollinger in the March issue of the *American Journal of Surgery* (**19**:411, 1933) has the following formula: 6 cc. of alcohol, 3 cc. of chloroform, 1 cc. of glacial acetic acid and 1 Gm. of ferric chloride.

We have all heard about caustics, as Dr. Roy brought out, but the trouble of using a caustic is that it seals off the entry and may not reach all the way. This formula was developed by these men in their work on cysts of the brain. They injected the solution in an effort to fix the cyst and then curet out what they could and dissect out what they could. They had considerable success. The experimental work was done on dogs. They modified the solution in order to make it fit their needs better.

Then Dr. Cutler of Harvard used it in this work and reported two or three cases. One hazard is that it burns badly if it gets on the skin or the epithelium. My use of it convinced me that if I employ it again I shall want to use some stain with it. It is somewhat difficult to see whether you really get through to the other end. Of course, if you put your escharotic in and it fails to go through to the other end, you have sealed off the half on which you have applied the caustic and have failed to reach the other half; this is what should be avoided. It should be applied all the way. If the escharotic goes through to the other end, then you must see it come through. I think that a stain can be had, such as methylthionine chloride, U. S. P. (methylene blue) or some other dye, which can be added to the caustic and with which one will be able to tell when the escharotic comes through. Of course, one must be very careful as the escharotic goes through so that it will not burn the other end.

Cutler and Zollinger recommend copious smearing with ointment, but in our cases we smeared the neck with ointment, and even then got a little burn on the outside. It does not seem to upset the patient. One little fellow went around the ward without any pain or fever, and we are sending him home.

I think the use of this solution will be more in a small, long cyst or tract than in a large, old chronic cyst, but that I cannot say. At least I think that we shall succeed, unless we fail to get the caustic to the extreme upper margin.

As regards surgical intervention, we all use some stain to try to color the tract as we work. Dr. Harris spoke about going up through in a thyroglossal cyst or duct. That, I think, is absolutely essential. I remember hearing a discussion years ago by Dr. Stein, I think of Chicago, in which he said that in most of the cases if you went as far as the hyoid bone, that was sufficient. In two cases I had to go through the hyoid bone, split it and go on up; finally the fistula passed into the base of the tongue and began dividing and was dendritic in nature. After it divided into three or four tracts, I lost it. But it progressed upward and evidently divided; instead of going by one point, it went by three or four points and so drained through the base of the tongue.

I was very interested in Dr. Figi's report of heredity. I suspected that history in ten cases.

FURTHER OBSERVATIONS ON THE CHEMICAL COMPOSITION OF SINUS PUS. DR. SIDNEY ISRAEL and DR. H. O. NICHOLAS, Houston, Texas.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. LEE WALLACE DEAN, St. Louis: When I began the practice of laryngology, I was told that the presence of cholesterol indicated that one was dealing with a cyst and not with a chronic suppuration. The textbooks that I used and some of the textbooks of today say that when cholesterol is found in aspirated fluid from the cavity adjacent to the nose, there is a dental cyst or a dentigerous cyst. But in the discharge from chronic suppuration of the sinuses, one can find cholesterol crystals just the same as in the fluid which is obtained from one of these cysts. The finding of cholesterol is not indicative of a cyst.

If I may digress a little, I should say that we have secured more information from the study of the chemistry of the discharge from the ear, from the practical standpoint, than from that of the chemistry of the discharge from the sinus. I have proved, I think, with Miss Burmeister and Miss Fenstein, that the presence of cholesterol crystals plus fatty degenerative epithelial cells has nothing to do with the diagnosis of cholesteatoma.

We have found discharges from the ear without cholesterol in cases in which enormous cholesteatomas have been found at operation. The amount of cholesterol which is present in a discharge from the ear depends on the accessibility of the cholesteatoma. If it happens to be in a peripheral part of the mastoid, there is little cholesterol in the discharge from the ear. There is just as much cholesterol in ordinary cerumen as there is in cholesteatoma.

In otitis externa without perforation of the drum head one can find the thing which even such a good textbook as Kerrison's says is indication of the need for a radical operation on the mastoid. In Politzer's, Kerrison's and other outstanding textbooks of otology, one finds the statement that the presence of cholesterol crystals plus fatty degenerative epithelial cells indicates the presence of cholesteatoma and the need of a radical operation. In otitis externa cholesterol crystals and fatty degenerative epithelial cells can be found in large amounts, and naturally there is no involvement of the middle ear.

In otitis media of two weeks' duration cholesterol and fatty degenerative epithelial cells can be found.

In connection with the nasal discharge, we have been interested in the p_H, the viscosity and the nitrogen content. In uncomplicated allergic rhinitis the nasal discharge is always alkaline. Again, in connection with the presenters' observations, in allergic rhinitis in which there are a typical nasal hydrorrhea and an abundance of nasal discharge, it is axiomatic, I think, that one should find a smaller amount of calcium than in a discharge from the nose of small amount.

When a patient comes to the clinic and drinks a glassful of water and the water runs out of the nose, as it does in nasal hydrorrhea, and one can collect an ounce in a few minutes, if that patient has an infection of the sinuses with the production of larger amounts of calcium, I do not think that one would expect to find so much calcium in the nasal discharge.

The discharge in allergic rhinitis is alkaline. In some of the patients an infection has developed after the allergic rhinitis, and with the appearance of the infection the nasal discharge has become acid.

Here is something that is entirely new, and this is Miss Burmeister's observation. We all know that in allergic rhinitis the presence of eosinophils is an exceedingly important diagnostic point. In every case of allergic rhinitis that Miss Burmeister has studied in which there have been in the discharge an enormous number of eosinophils, the moment the reaction of that fluid changed from alkaline to acid, the eosinophils disappeared; this was something entirely new to me. We hastened to the laboratory and took our stain, and we secured a non-nasal discharge filled with eosinophils; we made it acid to see whether the acidity interfered with the staining of the eosinophils. It had no effect whatsoever.

In studying the chemistry and the cytology of the nasal discharge and the discharge from the ear in known cases of allergy, in only one instance have we ever found eosinophils in the discharge from the ear. I mean to say that in a case of allergic rhinitis with an abundance of eosinophils in the nasopharynx and

in the nose, there are a reddening and a bulging of the drum head; the drum head is cut, and the boys in the clinic say: "Here you are going to find these eosinophils. This is the case where you find them." But they have never been found, that is, in increased numbers. I should not mention that fact, because Miss Fenstein is now starting a study of the chemistry of the discharge from the ear to see if, perchance, in infections of the middle ear the fluid is always acid; that may have a bearing on the eosinophils.

SUPPURATION OF THE PETROSAL PYRAMID: I. ROENTGENOLOGIC PROBLEMS. DR. HENRY K. TAYLOR, New York. II. PROBLEMS CONCERNED WITH ANATOMY, PATHOLOGY AND SURGICAL THERAPY. DR. SAMUEL J. KOPETZKY, New York.

The petrosal pyramid may consist of either pneumatic or diploic cells, either type of which may be recognized on roentgenograms. True petrositis must occur in the pneumatic type, producing a coalescent infection with diminished aeration and increased density, resulting from subsequent osteitis.

Confusion has been prevalent surrounding the symptoms of so-called Gradenigo's syndrome. As originally described, these symptoms did not indicate petrositis, which may be present without paralysis of the sixth nerve. All cases of true petrositis occur in the pneumatized temporal bone, and the more cellular the petrous pyramid, the more evident is the tendency to suppuration. In these cases the pyramid is developed by pneumatization just the same as the mastoid process. It is true osteitis, not osteomyelitis, since there is no medullary cavity in pneumatized bone. Most of these cases have cells extending out into the zygoma. The infections of the mastoid and the middle ear usually clear up, and then after a quiescent period profuse otorrhea marks the beginning of the petrositis. The acute cases, if untreated, may lead to meningitis. The chronic cases lead to a chronic fistulous discharge without meningitis. Few, if any, of the patients recover without some form of surgical intervention. In many of them the condition is accompanied by pain in the eye, on the side of the lesion, especially at night, and they often exhibit a low grade fever. In addition there may be drowsiness, vomiting and irregular nystagmus. The cochlear and vestibular functions are usually normal. At this stage of the condition a roentgenogram will show changes if compared with the previous roentgenogram of the more normal state. These symptoms may, if neglected, disappear during a quiescent period, during which, however, the profuse discharge persists with the sudden development of generalized meningitis. In the acute case in which operation is not performed, the patient does not usually recover without surgical intervention. In many ways the chronic cases simulate the acute ones. The headache is, however, more generalized. Roentgen study affords the best means of diagnosis, and the difference between these and the acute cases is often only one of degree. The chronic cases usually end as chronic otitis media. Various operative procedures have been advocated for reaching the petrous pyramid: 1. A modified radical operation on the mastoid, in general not adequate to reach the lesion. 2. An operation which enters the pyramid through the arch of the superior semicircular canal. This is inadequate for cases in which there is no fistulous tract. 3. A radical operation on the mastoid which exposes the glenoid fossa by removal of the anterior bony canal wall and permits delivery of the carotid artery out of its bed. 4. Unlocking of the petrous pyramid with removal of the anterior and posterior buttresses. This procedure enters the pyramid at the upper portion with resulting bad drainage. 5. A procedure advocated by Dr. Kopetzky, consisting of radical operation on the mastoid, the taking down of the anterior canal without exposing the glenoid capsule, removal of the tensor tympani muscle and the use of a burr at the roof of the eustachian tube at an angle of 23 degrees from the external auditory canal wall, thus avoiding damage to the cochlea. Since the procedure evacuates pus from the infected cells as soon as they are opened, it results in no deformity and permits rapid operation and good drainage. There is, however, some risk of danger to the carotid artery.

DISCUSSION

DR. H. I. LILLIE, Rochester, Minn.. It is difficult to discuss such a comprehensive and authoritative exposition of the pathologic processes which involve the petrous pyramid. It is my opinion that the clinical entity is well established and readily recognizable. With reference to the pain behind the eyes, van Voorthuysen believes that it is due, not to direct involvement of the ganglion or to traction on the ophthalmic nerve by an inflammatory swelling of Meckel's sheath, but to irritation of the sensory nerve endings of the recurrent ophthalmicus of Arnold, arising from the lateral undersurface of the ophthalmic nerve before the deep ocular branches separate from it, and passing over the tip of the pyramid toward the occiput. It innervates the bony substance of the petrosa.

Relief may be obtained by adapting an adequate operative technic to the pathologic process encountered. For closed empyema, for which Dr. Kopetzky says his technic is particularly adaptable, no more satisfactory method has been suggested. The three patients whom I have observed did not require the radical exposure of the mastoid because the discharge was found to come from a tract in the epitympanic region leading to the apex, where pus had collected. Exposure of the epitympanic region was carried out in a manner similar to that in which the root of the zygoma is exposed in a zygomatic abscess. This can be done without disturbing the structures of the middle ear. Secretion in the middle ear is occasionally observed following operation on the gasserian ganglion because certain of the zygomatic cells have been opened in exposing this same apical region.

If the infection in the apex occurs as the result of extension from the lower tubal cells, it would be necessary to approach the apex from a region that only a radical operation would expose. Certain aural surgeons do not use drills, and if exposure may be had by other tools, I can see no objection to their use. Kuster's suggestion that the surgeon "open up the bone freely so that it can be readily scrutinized to clear away all disease and to expose fully the source of suppuration so that the pus is nowhere checked in its outflow" has made a strong impression on me.

In my opinion, the elucidation of this clinical syndrome and its surgical relief have been the outstanding advances of the last decade in otologic surgery.

DR. C. STEWART NASH, Rochester, N. Y.: Dr. Kopetzky divides suppurations of the petrous apex into two types: first, the acute case, in which, if unrelieved, acute generalized meningitis develops and the patient dies; second, the chronic case, in which a fistula develops and which results in either a spontaneous cure or a chronic discharge from the ear.

Surgical treatment of the petrous apex offers patients with the first type, that is, acute suppuration, the opportunity to get well. In my experience, these patients all die. It offers to patients with the second type, that is, chronic suppuration, the opportunity to have a dry ear. If you have not already taken the time to examine the x-ray pictures in the back of the room, taken by Dr. Taylor or Dr. Kopetzky, you should look at them. If you do not know the history back of some of the pictures, you should inquire.

One of these films—I believe it is the middle section labeled "J. B., 21 years of age"—represents the type of case in which in my experience the patient always dies. This patient is perfectly well today. That was the case Dr. Taylor showed in which the iodized poppy seed oil 40 per cent settled in the cranial vault through a perforation in the petrous apex. This work is pioneer surgery. The work these men have done is a definite step in advance in otologic surgery.

The question is what is to be the attitude of the individual otologist toward surgical intervention on the petrous apex. I believe that this group of otologists, or any group of otologists, at the present time may be divided into three classes, according to their attitude toward such surgical intervention: First, there is the person who under no circumstances would operate on a petrous apex. Second, there is the person who would operate on a petrous apex providing he could

give himself a sufficient amount of exposure so that it would be a safe method. At the time of this writing he would probably follow a method devised according to the ideas of Dr. Eagleton. Third, there is the person who has no fear in working in a restricted field but has absolute confidence in his ability in dead-reckoning and in direction-finding.

If you have these qualifications, my belief is that the Kopetzky-Almour method is the method of choice, because it gives an approach to the petrous apex at the easiest access, at its point of greatest dependency. Then, assuming all of these premises, I believe that this operation is the most conservative from the point of view of the patient.

DR. J. GORDON WILSON, Chicago: I was pleased to see in the announcement of this discussion that Dr. Kopetzky proposed to introduce this subject with a study of the anatomy of the petrous bone. I presumed that he did so because he regarded this as essential to an understanding of its pathology and not merely a preliminary to the operative technic which he has so well carried out. I believe that a consideration of the complicated structure of this bone will help to a better understanding of its pathology and suggest an answer to some puzzling questions which arise during the course of its infection.

My remarks will be confined to the apex of the bone, to its anatomy, and only briefly and incidentally to its pathology. Whatever I say must not be construed as belittling the frequency of suppuration at the tip, the importance of roentgenography or the necessity at times of operation.

There are two noteworthy changes in the petrous bone related to and dependent on the growth of the brain—a change in size and a change in position. Both progress up to and even beyond puberty, to a still undetermined age. The change comes about from the position of the bones at the base of the skull, jutting out at an angle from the basioccipital and sphenoid bones. But it is not the entire petrous bone which grows. The membranous labyrinth after birth is not altered materially in size, if at all; its postfetal length remains unchanged in the child and in the adult. The cartilaginous capsule of the labyrinth, later the bony capsule, which surrounds the membranous labyrinth, does not materially increase in size after birth, and this is particularly true of the part around the cochlea. However, it is known that the petrous bone does alter in size, form and position to conform to the growth of the cranium. From measurements I have found, its length in the adult is on the average one and one-half times as great as that of the bone in the infant, and its width is virtually doubled. Thus, while the essential portion of the labyrinth and its immediate surroundings remain unaltered after birth, the whole bone undergoes a marked increase in size, and this increase is due to bone deposited under the periosteum and to an increase in size of the spaces which contain the red marrow cells at the tip, an increase in the trabeculae and an increase in marrow cells. In this there is a large, and certainly during early life an increasing, localization of myeloid elements, an activity associated with growth. It is agreed that myeloid tissue belongs to the reticulo-endothelial system, and that it has not only important metabolic functions but also important defensive functions against micro-organisms. It is known that all general pathologic processes immediately affect the bone marrow, and that in most general infections, for instance, during suppuration, the marrow is stimulated. I believe that marrow is equipped to deal with an infection reaching its tissue. What factors determine the breakdown of this defense I do not know, but I feel safe in saying that most pathologists agree that a slight or passing infection may well be dealt with without forming a pus cavity.

My microscopic examinations have shown: 1. Congestion of the tip is very common, varying in degree, even if the ear is not diseased. It is present in every one of my cases following meningitis whether or not the middle ear or the labyrinth is involved. 2. If infection is prolonged or perhaps not severe, one sees granulation tissue in the marrow spaces. 3. In the child the infection is vascular. 4. I have seen cases in which the suppuration from the tip has burst through the cortex, either under the dura or toward the middle ear.

Regarding the question of pneumatic cells at the apex, it is asserted that "there have been frequent demonstrations of almost complete pneumatization of the petrous tip. Occasionally single large cells are present which practically hollow out the entire apex" (Metzer). Or, again, "It cannot be denied that the majority of petrous pyramid infections which reach the apex do so by direct extension through the (? pneumatic) cells—a layer of cells extend along the eustachian tube from the tympanum to the petrous tip" (Friesner and Druss).

I have examined in sections a large number of petrous bones of children and have not found a typical pneumatic cell at the tip. I have also examined sections of a number of petrous bones of adults and have found pneumatic cells associated with the eustachian tube, but none in which these pneumatic spaces encroached on the marrow cells of the apex. I do not say that pneumatic cells may not exist in that region. It is notoriously difficult to prove a negative, but I have never found them.

DR. ELMER W. HAGENS, Chicago: My experience with temporal bones has been somewhat different from Dr. Wilson's. I have studied perhaps more newborn babies than adults. I have a small series of adult temporal bones which I feel show the anatomy as Dr. Kopetzky has outlined it. They show definite pneumatization in the anterior part of the petrous bone. These cells are variously developed in the various temporal bones, and the anatomy seems to differ a great deal, from absence of pneumatization to very extensive pneumatization.

I always find a certain amount of marrow anteriorly, anterior to the cochlea, just as Dr. Wilson showed, but, in addition, one can find pneumatic spaces extending very close to the marrow spaces. In one section there are a large pneumatic cell, then a marrow space and then a carotid artery. So I feel that in the type of case as qualified by Dr. Kopetzky, one can very well have the condition that he has outlined, and when cases of that particular type are chosen, the operation should be indicated and should be successful.

DR. FREDERICK M. LAW, New York: In discussing this valuable paper I shall confine my remarks strictly to the roentgenologic diagnosis of inflammatory changes in the petrous bone.

While I fully appreciate the thorough work done by Dr. Taylor and Dr. Kopetzky and value their conclusions highly, I desire to utter a word of caution to the roentgenologist or the surgeon who has not done extensive work in this narrow field and who is liable to make a misinterpretation from some of the films submitted for diagnosis.

The danger lies in making a diagnosis of petrositis or of suppuration of the apex of the pyramid from the appearance of the roentgen films alone.

While I am a dyed-in-the-wool roentgenologist, I cannot forget that I was an otolaryngologist and must consider the clinical evidence. I believe that in the type of case under discussion one must give the clinical evidence 75 per cent of the glory and the roentgenogram the remaining 25 per cent and consider the latter as confirmatory.

The petrous pyramid is the most difficult portion of the skull to show clearly on a roentgenogram, and as the changes which are described are sometimes faint and indistinct, one must have some basis for comparison in order to be sure of one's opinion.

Routine films made in cases of sinusitis and mastoiditis without any petrous involvement have shown many differences in the development and the character of the cells in the two petrous bones. Granted that both sides should be similar, as is usually the case with the pneumatic mastoids, it is reasonable to suppose that when there is a difference in the development or character of the walls of the cells this difference is the result of some previously existing inflammation or of the present attack. This difference is shown in a pneumatized petrosa as a hazy, fuzzy outline of the walls of the cells and a decreased density of the bone or actual loss of structure of the bone from necrosis. There may also be an increased density from an osteitic thickening.

Sections made at autopsies by Dr. Edmund P. Fowler have shown evidence of inflammatory exudate in the petrous cells in cases without a history of disease of the mastoid.

It must be concluded that in all cases of involvement of the mastoid the inflammation extends into the petrous cells.

When it comes to making a diagnosis of change in the petrous bone from the films, one must consider the changes that are observed in so many cases without mastoiditis. This is where my word of caution enters. Be extremely cautious in attempting to make an interpretation of petrositis from a single set of films.

If there is definite evidence of suppuration at the petrous apex and a set of films shows a normal outline on the good side and an unmistakable absence of outline on the affected side, one may conclude that necrosis is present, but when the clinical evidence is not conclusive, one must have a previous set of films for comparison. It is also essential that the films be made from two angles and also stereoscopically, with good density. Unless the films are technically perfect, they are worse than useless.

In all of my examinations of the mastoid or the accessory sinuses, I make routine films of the petrous bone in order to have a record on which a comparison may be based if complications should develop. A thin and transparent marrow bone tip of the petrosa may be mistaken for necrosis.

In the examination of hundreds of Dr. Fowler's cases of progressive deafness, we have found such distinct differences in the appearance of the petrous bone that I am impressed with necessity for extreme care in the interpretation of changes in the petrous bone unless there are definite clinical evidence and films for comparison.

Dr. Taylor has stated that his cases are those which showed actual absorption of bone at the apex of the pyramid, but I am afraid some of you may leave here and subsequently ask a roentgenologist to find evidence of inflammation of the petrous bone without destruction of bone; hence, my words of caution about depending on the roentgen films alone for a diagnosis.

DR. LOUIS K. GUGGENHEIM, St. Louis: I am sure that I speak for every one present in saying that we have thoroughly enjoyed Dr. Kopetzky's classic presentation of this most important subject and Dr. Taylor's excellent demonstration of x-ray pictures.

My reason for arising is to present for consideration a new treatment for petrositis, and to report three cases in which recovery took place. The treatment is nonsurgical.

A boy of 18 was operated on in New York City for simple mastoiditis; he did not recover. He was not in Dr. Kopetzky's service. He came to St. Louis and entered our hospital. The physician in charge operated again (this was many months later); he did further exenteration. The boy did not recover. He had then lost weight to 120 pounds (54.4 Kg.). My service started at that time. When I examined this youth, he had intense pain above his left eye. He complained so bitterly that the nurses were disgusted, because they had diagnosed his case as one of psychoneurosis. He had a mother fixation. The mother daily wept over him and petted and kissed him. The patient was suffering intensely with pain above his left eye. He had fever and paralysis of the external rectus muscle on the left side. A diagnosis of petrositis was made. Roentgenograms were taken. One of the pictures is on the board in the back of the room. He had all the classic symptoms of petrositis.

I have never had the temerity to do the Kopetzky-Almour operation, which I think is most ingenious and most excellent but most dangerous; that is, it would be in my hands at the present time, because I have not done it on a cadaver. The patient was taken to the operating room for the third time, and the petrous pyramid was exposed by removal of the tegmen tympani, the dura being elevated. There was no extradural abscess. The petrous bone was apparently perfectly solid. I made an incision posteriorly, exposed the other surface of the petrous pyramid and found nothing, so decided to stop.

Thereafter, the following treatment was carried out; that was the first time it has been tried: The patient was treated hourly with massive suction. Paralysis of the rectus muscle on the right side developed. He finally recovered. The paralysis disappeared. I thought that I was dealing with basilar meningitis. The ear became dry, and the patient now weighs 175 pounds (79.4 Kg.).

There were two other patients who were not operated on. They merely underwent simple exenteration of the mastoid. There followed in both cases paralysis of the external rectus muscle, fever and pain in the eye, with the classic picture shown in the roentgenogram in the back of the room. The patients were simply treated with 95 per cent solution of alcohol and massive suction of the external canal hourly. The three patients were all put back in the hospital, and they all made a recovery. All three had paralysis, fever and pain in the eye; two patients recovered completely, and the third one is practically well.

Dr. Samuel J. Kopetzky, New York: We have not the time to exhaust this topic. I know it will be a subject that will be before many meetings before it is entirely thrashed out. My object was to set certain fundamentals which are not in dispute but about which there was considerable confusion and misunderstanding. I thank the members for the courtesy with which they have taken up this topic and discussed it.

Dr. Lillie, I will say that no one but (I was going to say an amateur) a beginner would lay down any general rule applicable to any lesion, because the lesion differs with the individual patient and with the underlying anatomy.

There is evidence in the presentations of Dr. Gordon Wilson and Dr. Hagens that the anatomy differs. With the different anatomy there is a different pathologic anatomy, and hence there is no fixed rule to which all cases can be submitted. Medicine is not as simple as all that.

Dr. Gordon Wilson brought out the fact that the petrous pyramid is a growing bone, and a growing tissue is subject to pathologic assault more easily than a bone that is set and fixed. I cannot enter at the present time into a discussion of Dr. Gordon Wilson's findings. I know that his observations are true, and I shall have to find an explanation to correlate, to fit into the mosaic of this picture, his findings. Nevertheless, when pus develops, irrespective of the underlying bone marrow or bone cells lined with epithelium or broken-down defense marrow, if one has a means of recognizing it in situ and of evacuating it, I think one should use that means.

I hold to Fischer's fundamental factor in the differentiation between pneumatized and nonpneumatized cells, the presence or absence of the haversian system and of Volkmann's canal system. They are not present in pneumatized bone.

I do not say that all patients need to be operated on. The patients who have acute and encapsulated cases and are threatened with meningitis should be operated on. We have presented a way of evaluating the cases. Fistulous tracts can be evacuated; they can be cleaned and drained. They can be treated as Guggenheim treated them by mass suction. Mastoiditis can always be treated by putting a small hole somewhere in the mastoid and applying mass suction; eventually the ears become dry. We played with that treatment years ago. We do not consider it good technic nowadays.

Regarding Dr. Law's statement transmitted by Dr. Harris, emphatically, the clinical diagnosis here, as in most other instances, is predominant over any laboratory aid whatsoever. The laboratory is corroborative; it is not determinative. Surely at autopsy you and others have found pus in the tip cells without petrositis.

In the early days, when I used to give a course in surgery in the morgue, patients who died never had mastoiditis. When we opened the mastoids in demonstrating a surgical technic, we found that a certain percentage of patients had a purulent secretion in these cells. So far as we could find in going back in the history at Bellevue, they had never complained of mastoiditis. In other words, a lesion must be developed beyond a certain degree before the patient reacts to it.

In all cases of fistula in which we have cleaned and drained the tracts, the patients have gotten well. All of the patients with encapsulations on whom we

have operated have recovered. The only patient (one of Dr. Nash's) that we have had so far in whom the cyst ruptured intracranially recovered. Whenever the encapsulations have ruptured and the rupture has spread over the tentorium, over the occipital lobe of the cerebrum and into the sulcus habitum, we have not been able to cure the patients. Altogether I have had twenty-five cases. We have accepted twenty-five cases. Since the original publication, we have seen a couple of hundred. People have said, "Petrositis with or without equivocal pictures." Do not make the mistake of thinking that the disease is more prevalent today than it used to be.

ELECTROCOAGULATION OF THE CAVERNOUS SINUS TO PREVENT THROMBOSIS: REPORT OF A SUCCESSFUL CASE FOLLOWING CARBUNCLE OF THE NOSE. DR. MERVIN C. MYERSON, New York, and DR. E. JEFFERSON BROWDER, Brooklyn.

The considerations in this paper relate particularly to bacterial thrombophlebitis of the cavernous sinus which results from extension of infection by way of the ophthalmic veins. The anatomic peculiarities of the sinus, its immediate ramifications, the difficulty of early recognition of its involvement, its relative inaccessibility and the knowledge that bacterial meningitis may develop sooner or later are all obstacles to the surgeon who attempts to prevent the disease.

The various routes of surgical attack that have been advocated up to the present time are reviewed. All of the procedures which have been advocated have had as their objective incision and drainage of the cavernous sinus. One questions the rationale of incision and drainage of a thrombophlebitic vein in an effort to limit the extent of such a pathologic process or to eradicate it.

Cases illustrating a surgical attempt to cure involvement of the cavernous sinus are reviewed and commented on. It is stressed that the method that will cure must be one that will limit the infection to the orbital cavity by interposing a sterile clot as a barrier.

A case is recorded in which a man of 23 had a carbuncle of the right side of the tip of his nose for three days, when he had a slight chill, and his right eye became proptosed and the conjunctiva chemotic. The orbital movements were limited, and he had what appeared to be an early picture of cavernous sinus thrombophlebitis. Blood cultures taken at this time revealed Staphylococcus aureus. A culture from the carbuncle and the incised angular vein yielded the same organism.

Operation was performed as follows: The right temporal lobe was exposed and the dura split after ligating the middle meningeal artery. The temporal lobe was elevated intradurally and the cavernous sinus exposed. A coagulating needle connected with a powerful Bovie unit was inserted into the anterior end of the cavernous sinus, and the lining of the sinus was obliterated. The postoperative course was marked by severe suppuration of the orbital contents, and numerous incisions were required for the evacuation of pus. At the end of sixty-six days the patient was discharged from the hospital as well, but his right eye was completely blind.

This procedure is contraindicated in the presence of clinical evidence of bilateral orbital infection. When the cavernous sinus is involved by a thrombophlebitic process through a pathway other than that of the ophthalmic veins, it is our opinion that this procedure is contraindicated. In instances in which the wall of the cavernous sinus is infected, it is possible that electrocoagulation might cause diffuse leptomeningitis. Further experience is necessary to establish firmly the correctness of this statement.

DISCUSSION

DR. HARRIS P. MOSHER, Boston: I congratulate the presenter and his colleague on their courage in trying this procedure. The cranial route to the cavernous sinus has much to be said in its favor. It is not justifiable unless the operator has perfected his technic in intracranial operating. We were on the

point of trying this operation in a recent case, but the combined judgment of the consultants was that the case in question had advanced too far to make operation justifiable.

The presenter starts with the statement that "one questions the rationale of incision and drainage of a thrombophlebitic vein in an effort to limit the extent of a pathologic process or to eradicate it." I do not agree. That attitude would do away with tying the jugular vein in the neck, opening the lateral sinus or evacuating a septic focus wherever found. In other words, it is in entire opposition to the oldest surgical principle.

Obliteration of the lining of the cavernous sinus seems to have a disastrous effect on the eye, that is, on the sight. In the presenter's case it did not prevent mestastases in the chest.

The cranial route is most direct. The route of Langworthy is too complicated anatomically. The oblique shot of Luc does not appeal to me. I like a straight shot when possible. Of necessity, there are too many oblique shots in nasal operations as it is.

I should like to say a word more about the direct orbital route. I should like to come back to the orbital route because of late years I have acquired a little information which makes that route appeal to me a bit more perhaps than it did in the beginning. The chief objection to the orbital route is the removal of the eye. In most cases the sight of the eye will be lost. If you use the orbital route, it is either a dead eye or a dead patient. It is only of late years, after I had seen the results of autopsy in three cases of cavernous sinus thrombosis, that I realized how extensively the orbital contents were riddled with abscesses. The orbital contents were one mass of thrombosed veins and arteries and small abscesses. The sheath of the optic nerve was a bag of pus, and I saw one case of thrombosis of the central artery and vein of the retina. In other words, I wonder whether the orbital route is not indicated on this account—whether the orbit should not be cleaned out on general surgical principles. We had a recent case in which this was done, with recovery.

One usually thinks that edema of the lids means extensive thrombosis of the cavernous sinus. Why could it not be due at first to the extensive infection in the orbit itself? The more I think of it, the more I think it could be so and the more I think that the orbit should be cleaned out on general surgical principles, and not cleaned out to furnish an approach to the cavernous sinus.

The orbital route drains pus into the orbit and, with the orbit exenterated, drains the pus on the surface of the body rather than through the cranial cavity. Drainage by the orbital route is more in line with one's surgical instinct.

I agree with the presenter that when the second eye is involved operative treatment is useless. It is most discouraging how quickly the second eye becomes involved in so many cases.

The presenter said that tying the angular vein was advocated years ago, in 1910. It has always seemed to me that the angular vein is a very trivial vein to be associated with so much trouble, but it is. It occurs to me that in infection of the sinus from carbuncle of the lip or infection of the vestibule of the nose it might be worth while to make a routine procedure of tying off the angular vein or, what is simpler, of using the procedure or the instrument advocated here, blocking it off by diathermy.

DR. O. JASON DIXON, Kansas City, Mo.: I believe that in addition to the study of the anatomy of this very inaccessible sinus, we must go back a step further and study the physiology and pathology. The formation of the thrombus, which is nature's ligation, the means of separation of the infected field from the circulating blood, is a normal physiologic process. It is only when the thrombus becomes infected and constitutes a pathologic picture that we get into trouble. I think that it is a very fine decision to determine when to interfere and when not to interfere. There is one thing in our favor in the way of interfering, in that we know that practically all of the patients will die anyhow.

I have recently seen two cases. So far as I could determine clinically from photographs and every other study, they were typical cases of cavernous sinus thrombosis. The patients have both recovered. In one of them I attempted to prevent the extension of the infection into the lung by ligation of the internal jugular vein, which was thrombosed secondarily in the neck. This started from an infection of the throat. This may sound against a principle I have been preaching.

The other patient had an orbital abscess, blood in his sputum and hemiplegia. Then osteomyelitis of the skull developed. He later had convulsions. Any one of these signs seemed enough to kill the patient, but he eventually recovered. To predict what happened in this patient, unless death occurs as a result of some other cause and a complete necropsy is performed, would be, I think, in the realm of theory and speculation.

I believe, as Dr. Mosher stated, that we must pay more attention to the orbital infection as the active focus which is still feeding the thrombus which nature has thrown down as a physiologic plug and reinfected. If the infection in the cavernous sinus could be treated without the development of meningitis or diffuse septicemia, with a septic infarct in the lung and all through the body, the patients would have a chance. I agree that when the other eye is involved, the condition is hopeless.

To my surprise, in the last few years I have found that the other eye is not necessarily involved, even when the patient dies, and also that this occurrence does not follow any set rule. Chemosis of the eye and bulging and all of the classic signs by no means indicate positively that the patient has had or will have cavernous sinus thrombosis. I think that we should try to treat the thrombosis, the extensive plug, the obstacle ahead of the progress of the infection, if we can do so safely without carrying infection into the new plug.

DR. MERVIN C. MYERSON, New York: I appreciate the discussion of Dr. Mosher and Dr. Dixon, but I still have to stand on my conviction that the procedures that have been advocated, or those that have involved incision and drainage of the cavernous sinus, do not accomplish what is necessary to prevent the advance of an infection.

With regard to using the lateral sinus as an analogy, I should call attention to the fact that when we encounter an infected thrombus in the lateral sinus, we do not incise and drain it, but we exenterate that sinus, which is just what we are advocating for the cavernous sinus. I should also say that if there is an infected thrombus in the cavernous sinus, I am not so sure that electrocoagulation would save the patient.

In the matter of metastases in this particular case, we believe—we may be wrong—that the metastases could have occurred before operation; however, they might have occurred after the operation.

The orbital route has a great deal to commend it. In discussing the orbital route with Dr. Browder, the co-author of this paper, I wanted to include in the paper the fact that we should advocate electrocoagulation in the cavernous sinus, and then immediately afterward evisceration of the orbit, but Dr. Browder said, "You may get into trouble operating in so infected a field, and you had better not mention that in your paper because it is open to considerable question."

Dr. Dixon gave us a very scholarly discussion, but he contradicts himself in places. He speaks of a very inaccessible sinus, and I am sure you will agree with me that in the hands of one who knows his field, elevating the temporal lobe will give a very accessible sinus and a perfect visualization.

As to the question of theory and speculation as to whether patients are going to get well, I should say this: If I have a carbuncle of the nose and chemosis and proptosis, if I have had a slight chill and if I do not look good to you, I wish you would perform this procedure on me and not theorize and speculate on the probable outcome.

Our intention in presenting this case and this paper was to advocate getting ahead of the infection before it reaches the cavernous sinus. Get at the cavernous sinus while it is still normal and interpose an aseptic clot, if you will, which will prevent the extension of that infection into the intracranial structure.

SARCOMA OF THE LARYNX: REPORT OF FOUR CASES. DR. FREDERICK A. FIGI, Rochester, Minn.

This article was published in full in the July, 1933, issue of the ARCHIVES, p. 21.

DISCUSSION

DR. M. F. ARBUCKLE, St. Louis: Dr. Figi's paper has brought me to the realization that I have never seen, so far as I know, a well authenticated case of sarcoma of the larynx in my private practice. Among the last 276,651 admissions to the Washington University Dispensary and among the last 90,623 admissions to the Barnes Hospital, there has been, so far as I can find, only 1 case of sarcoma of the larynx, and this was a spindle cell sarcoma. I did not have an opportunity to see this case, but I am told that the patient died.

I had been accustomed to think of malignant tumors of the vocal cord as of epithelial origin, and I was, therefore, somewhat surprised at the statement that sarcoma may arise from the vocal cord. Of course, it is possible, and I think that this is a point well worth comment.

In thinking over my experience with laryngeal tumors of questionable nature, I recalled 1 case which I examined two or three years ago in the Barnes Hospital. In checking over the records of this case, I found that the same difficulty was experienced by the pathologists as has been mentioned by Dr. Figi; namely, the diagnosis was carcinosarcoma, with a note by the professor of pathology that it was impossible for him to make up his mind whether the tumor could properly be classified either as carcinoma or as sarcoma. I have always been accustomed to think of sarcoma as a highly malignant type of tumor with early metastases and little possibility of cure, and it is with great pleasure that I have learned from Dr. Figi of my mistake in this direction.

I should like to place emphasis on Dr. Figi's recommendation that the diagnosis of tumor of the larynx should be accepted only when it has been made by a competent pathologist who has had special experience in the study of tumors from this region. This remark is prompted by the fact that many people do not seem to realize the tragedy which is immediately in the offing when a mistake is made in either direction in the study of tumors such as these.

I must confess that I am astonished and delighted at the statements concerning the apparent ease with which Dr. Figi deals with sarcoma of the larynx and secures such excellent results.

Dr. Figi has not mentioned dissection of the glands of the neck, and while I assume that it is his custom, as it has been mine, not to do dissection of the glands in cases of carcinoma of the larynx in which the disease is definitely and surely intrinsic, I should like an expression from him concerning his practice in regard to dissection of these glands in cases of sarcoma of the larynx which are definitely intrinsic.

I wish to express my appreciation to the essayist for his excellent review of the literature on this subject. Papers such as this encourage us to study and record the findings in our cases more carefully, and certainly Dr. Figi's remarks concerning the matter of prognosis and treatment lend encouragement.

DR. LEE M. HURD, New York: Some time ago I had a case of sarcoma in a woman, aged 45, which formed a ring just below the cricoid cartilage, at the junction of the cricoid cartilage and the first ring of the trachea. On looking at the larynx, a white ring was seen. I did a thyrotomy and shelled out the tumor, which Dr. Don White pronounced a sarcoma, I have forgotten what type. The patient did not recover. About three or four months later she had a recurrence in the antrum and orbital region, and she died of a cerebral metastasis.

DR. FREDERICK A. FIGI, Rochester, Minn.: Answering Dr. Arbuckle's query concerning the matter of metastasis in these cases, I think I can do no better than to read a statement in which I quote Butlin. His explanation of the unlikelihood of metastasis in these cases is as follows: "The sarcomas of these parts arise by proliferation of the cellular elements of the solid structures of the connective tissues. As the cells proliferate, the solid structures gradually swell, and the lymphatic vessels suffer a diminution in their caliber which proceeds to their complete obliteration and renders them incapable of transmitting the infecting material of the tumor." Slobodnik noted a definite periodicity in the growth of a tumor of this type, as regards both the entire tumor and its various lobules. Although the tumor in his case had remained practically stationary for about two years and did not appreciably change the size of the lumen of the glottis for a year, subsequently, during the course of three weeks, it grew very rapidly.

As regards the matter of dealing with the glands in cases of this type, we do nothing unless there is definite clinical evidence of involvement later, directing all attention to eradication of the primary lesion. In cases of carcinoma we treat the patients in the same manner. In cases of active laryngeal carcinoma with involved glands, I think there is, as a rule, little gained by block dissection, which would be necessary for effective treatment of the lymphatics and which, of course, must be supplemented with laryngectomy if the tumor is sufficiently extensive to necessitate this. So we do not accept for surgical treatment patients with extensive active laryngeal carcinoma with definite cervical metastasis.

In cases of low grade carcinomas we do primary dissection of the glands and follow this with laryngectomy.

A CLINICAL ANALYSIS OF ONE HUNDRED AND FIFTY CASES OF ALLERGY OF THE NOSE AND PARANASAL SINUSES. DR. FRENCH K. HANSEL, St. Louis.

This presentation was a lantern slide demonstration of a series of allergic cases with complete data as to their history and clinical findings.

DISCUSSION

DR. WILLIAM V. MULLIN, Cleveland: Dr. Hansel's meticulous study of allergy bespeaks the fact that he is a very good allergist. A man who is busy and carries on a large general practice in otolaryngology or in any other field can hardly do his own work on allergy. I believe that perhaps four or five new cases of allergy are all that any one man can possibly attend to in a day if he works them out right.

I believe that there is great benefit in having patients return within twenty-four hours after a test, to note the effect of any delayed reactions. Sometimes it helps to sift out the false positives. I should disagree with Dr. Hansel in only one thing; I do not like the term "nasal allergy." These people are all allergic and are potentially liable to have a reaction in any part of the body.

Analysis of eighty cases showed some form of allergy preceding the nasal symptoms. In thirty-three cases other manifestations accompanied the nasal symptoms. That is my reason for objection; you can hardly separate allergy of one part of the body.

Dr. Hansel told me that they allow him to play the part of a roving sentinel, as it were, and he can go to the pediatric department and examine patients and to the clinic for patients with asthma and examine them. Of course, that is a splendid combination. If that procedure could not be carried on and if you drew the very distinct line and talked about nasal allergy and allergy elsewhere, there might be a group in between in which some cases would be missed.

In getting the history, if the case seems to narrow down to being the seasonal type of pollinosis or hay fever, one should not stop there but should carry out the intradermal test of the skin, because treatment will be influenced very much if that is not done. In other words, if the patients have pollinosis and are also

sensitive to foods, better results will be obtained from treatment by elimination of the foods to which they are sensitive.

Dr. Hansel spoke of a great many of the irritants. I should like to add silk. That gives quite a good deal of bother, particularly in the case of shop-girls who work in that department. It is most unfortunate when people become sensitive to their working environment.

I am sure that Dr. Hansel would like to add to his paper the method that is used for getting nasal secretions for study of the eosinophils. Dr. Hansel makes these studies himself. He looks the secretions over many times before he happens to find the clump that contains the eosinophils which would help him to make the diagnosis. That is so different from the method of the average busy man, who reaches in, takes a smear and sends it to some one else; the report comes back "no eosinophils." Furthermore, if the secretions were taken only by smear on a cotton swab, the eosinophils would be so distorted that the examination would be futile.

In regard to foods, we think that there is great value in having these patients use food diaries, because it gets them interested in their own problem. These sensitive people are indifferent. They consult one, and want something done quickly. That is why they like to have surgical treatment.

Dr. Hansel says that only 50 per cent of his patients would remain for study and treatment for as long as three months. So if the patients can be made to keep food diaries, they will be a little more interested in their own problem and will help with it when they come back for check-up studies.

I was glad to hear Dr. Hansel say that surgical procedures are necessary in only a few cases. I am more and more surprised at the changes that take place in the edematous polyp when the cause for the allergy has been removed.

The morning I left to come here I saw a woman I had not seen for seven years. She certainly presented an allergic picture when I saw her at that time. I am sorry to say that she was not properly studied. She did have some obstruction in her nose, so I performed an operation on the septum. She tells me that that operation gave her considerable relief, and yet I do not know why it did, because when I saw her the morning I left home the picture in her nose—the edematous membrane, the filled membrane—was exactly the same as when I saw her seven years ago. She is definitely allergic. I believe that now she will get the result that she did not get when I saw her seven years ago.

I am full of admiration for Dr. Hansel's work.

DR. AUSTIN A. HAYDEN, Chicago: I should like to speak of one point in the paper. Of a group of 125 adult cases of nasal allergy, the family history was positive in 64.8 per cent. The dictum of Kipling that the female is more deadly than the male is verified by the statistics that Dr. Hansel has produced, the female being about 47 per cent potent in the transmission of an allergic tendency, and the male about 24 per cent. This is a matter to which too little attention, I think, has been paid by otolaryngologists as well as by general practitioners.

I have in mind a family in which the members on both sides were strongly allergic. Such an error in genetics should be studiously avoided, and such advice should come more often from laryngologists than it does.

The care of these patients and the persistence of the patients themselves are, as Dr. Hansel and Dr. Mullin have said, important matters in successful treatment.

The avoidance of surgical treatment, except when it is absolutely necessary, has been emphasized.

DR. FRENCH K. HANSEL, St. Louis: Dr. Mullin brought up the point of how much time it takes to work out these cases. In conducting a clinic of this nature, it is best not to try to handle too many cases. I handle about twelve cases in three hours, and I find that I can work very nicely with those cases. It gives me time to write a good history. It gives me a little time to spend with each patient. I might suggest for private practice that a certain day of the week be regularly set aside on which one can spend three or four hours and work

out the cases and handle them. I think it can be done successfully in that way, if one is able to arrange one's other material for other days.

The term "nasal allergy" is used for lack of something better. Perhaps we shall get around later to some definite terminology throughout the whole subject of allergy.

I am glad to hear Dr. Mullin emphasize that in cases of hay fever the study of foods should play an important part. He brought up the question of food diaries. We used these diaries from the start. As soon as the patient comes in we start him on a diary to determine the kinds and quantities of foods he eats. Often the diary suggests that certain foods cause symptoms.

Later we put the patients on the so-called Rowe type of diet, which is free from meat, wheat and eggs. We modify this diet according to the skin test and according to individual tests. This elimination diet removes all of the common foods, foods that are eaten every day, and puts the patient on the unusual foods. It is remarkable how quickly the condition clears in some of the patients.

Then we slowly add back the foods and note which ones produce symptoms. In some cases we have much more difficulty than in others. They are all individual problems.

The question was asked as to what percentage of nasal polyps are of allergic origin. There are two types of polyps: Allergic polyps are the result of the long continued edematous process; first there is simple edema and then hyperplasia, and eventually the edema becomes so marked that the tissues prolapse and a polyp is present. There are also polyps in cases of suppurative sinusitis in which the polyposis is secondary to the suppuration. Thus, in some cases, polyps are secondary to the allergy, and in others they are secondary to the suppuration. We also had cases in which the polyps were permanently of allergic origin and then infection was superimposed. So there are these three things to consider in determining whether a polyp is allergic.

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President: Prof. Gunnar Holmgren, Karolinska Mediko Kirurgiska Institut, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143, Groningen, The Netherlands.
Place: Stockholm, Sweden. Time: September 2-5, 1934.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. W. P. Wherry, 107 S. 17th St., Omaha.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: 1934.

American Bronchoscopic Society

President: Dr. W. F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. W. V. Mullin, 9204 Euclid Ave., Cleveland.

American Laryngological, Rhinological and Otological Society

President: Dr. J. W. Jervey, 101 Church St., Greenville, S. C.
Secretary: Dr. R. L. Loughran, Bridgewater, Conn.
Place: Charleston, S. C. Time: April 3-5, 1934.

SECTIONS:

Eastern.—Secretary: Dr. Vernon L. Bishop, Medical Arts Bldg., Rochester, N. Y.
Southern.—Secretary: Dr. Fletcher D. Woodward, P. O. Box, 1685, University, Va.
Middle.—Secretary: Dr. Frank J. Novak, Jr., 30 N. Michigan Ave., Chicago.
Mid-Western.—Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Western.—Secretary: Dr. Ralph F. Davis, Mayer Bldg., Portland, Ore.

American Otological Society

President: Dr. John R. Page, 127 E. 62nd St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.